L7 ANSWER 1 OF 2 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2001:181747 BIOSIS
DOCUMENT NUMBER: PREV200100181747
TITLE: **Niacin** plus simvastatin protect against atherosclerosis progression and clinical events in CAD patients with low HDLc and diabetes mellitus or impaired fasting glucose.
AUTHOR(S): Morse, Josiah S. (1); Brown, B. Greg; Zhao, Xue-Qiao; Fisher, Lloyd; Chait, Alan; Dowdy, Alice; Serafini, Leny; Huss-Frechette, Ellen; DeAngelis, Debbie; Frohllich, Jiri; Albers, John
CORPORATE SOURCE: (1) University of Washington, Seattle, WA USA
SOURCE: Journal of the American College of Cardiology, (February, 2001) Vol. 37, No. 2 Supplement A, pp. 262A. print.
Meeting Info.: 50th Annual Scientific Session of the American College of Cardiology Orlando, Florida, USA March 18-21, 2001
ISSN: 0735-1097.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L7 ANSWER 2 OF 2 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1993:288990 BIOSIS
DOCUMENT NUMBER: PREV199345007115
TITLE: The effect on **coronary artery stenosis** of intensive pharmacologic step therapy to improve LDL and HDL in patients with normal plasma lipid levels.
AUTHOR(S): Sacks, Frank M. (1); Pasternak, Richard C.; Gibson, C. Michael; Rosner, Bernard; Stone, Peter H.
CORPORATE SOURCE: (1) Brigham and Women's Hosp., Boston, MA USA
SOURCE: Circulation, (1992) Vol. 86, No. 4 SUPPL. 1, pp. I743.
Meeting Info.: 65th Scientific Sessions of the American Heart Association New Orleans, Louisiana, USA November 16-19, 1992
ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

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(FILE 'HOME' ENTERED AT 17:15:24 ON 16 JAN 2002)

FILE 'MEDLINE, BIOSIS' ENTERED AT 17:15:37 ON 16 JAN 2002
L1 3785 S CORONARY(W)ARTERY(W)STENOSIS
L2 5 S L1 AND FISH(W)OIL?
L3 4 DUP REM L2 (1 DUPLICATE REMOVED)
L4 14 S L1 AND (DIET OR (LOWER? OR REDUC?)(W)(CHOLESTEROL OR FAT)(W)I
L5 10 DUP REM L4 (4 DUPLICATES REMOVED)
L6 2 S L1 AND NIACIN
L7 2 DUP REM L6 (0 DUPLICATES REMOVED)

L9 ANSWER 1 OF 34 MEDLINE
ACCESSION NUMBER: 2001208108 MEDLINE
DOCUMENT NUMBER: 21153014 PubMed ID: 11231647
TITLE: Initial experience with a newer generation coronary stent.
AUTHOR: Manolis A S; Chiladakis J; Hahalis G; Agelopoulos G
CORPORATE SOURCE: Cardiology Section, Patras University, Rio, Patras, Greece.. asm@otenet.gr
SOURCE: JOURNAL OF INVASIVE CARDIOLOGY, (2001 Mar) 13 (3) 217-22. Journal code: BCZ; 8917477. ISSN: 1042-3931.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010521
Last Updated on STN: 20010521
Entered Medline: 20010517

AB BACKGROUND: Recently, several newer generation stents have become available promising to improve upon the results of coronary angioplasty (PTCA) with its attendant acute and chronic complications. The aim of this study was to prospectively review the results of a preliminary experience with the newer generation R stent in a series of 56 patients. METHODS: This study included 47 men and 9 women, aged 57 +/- 10 years, who presented with stable angina and/or positive exercise testing (n = 12), unstable angina (n = 42), or acute myocardial infarction (n = 2). A consistent approach by a single operator for implantation of the R stent (Orbus Inc., The Netherlands) included stent oversizing (by 0.5 mm) and high pressure (> 12--16 bar) deployment. Dilated vessels comprised the left anterior descending (n = 37) including the diagonal branch in 2 patients, the right coronary artery (n = 17), the left circumflex (n = 13), or a saphenous vein graft (n = 1). The mean left ventricular ejection fraction was 52 +/- 8% and the initial **coronary artery stenosis** was 85 +/- 8%. Stents were implanted for dissection and/or suboptimal PTCA result or electively. RESULTS: The procedure was successful in all 56 patients (100%). The residual stenosis was < 0--10%. Direct stenting without balloon predilation was performed in 21 patients. Single stents were used in 36 patients and > or = 2 stents in 20 patients. Abciximab (n = 22), eptifibatide (n = 8) or tirofiban (n = 1) was administered in 31 patients (55%). A stent-related complication was noted in one patient (stent misplacement). All patients were discharged alive without infarct or need for surgery. There were no events of subacute stent thrombosis; all patients received combined therapy with **aspirin** and clopidogrel, the latter for 1 month. In one patient who had received abciximab, severe thrombocytopenia (0 platelet count) was detected at 3 days after discharge but it was fully reversible with no sequelae. Over 5.2 +/- 2.8 (range, 1--11) months, there was one sudden death and two clinical restenoses; no other late complication occurred. CONCLUSION: Initial experience with 73 R stents in 56 patients and a consistent approach by a single operator of stent oversizing and high-pressure deployment resulted in high procedural success (100%), lack of stent thrombosis (0%), and a low stent-related complication rate (1.8%), while the design and profile of the R stent allowed for direct stenting in 37.5% of patients. One should be vigilant for the sporadic occurrence of severe thrombocytopenia with use of IIb/IIIa inhibitors.

L9 ANSWER 2 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2000:283143 BIOSIS
DOCUMENT NUMBER: PREV200000283143
TITLE: Unprotected left main coronary artery stenting: Immediate and medium-term outcomes of 140 elective procedures.
AUTHOR(S): Silvestri, Marc (1); Barragan, Paul; Sainsous, Joel; Bayet,

Gilles; Simeoni, Jean-Baptiste; Roquebert, Pierre-Olivier; Macaluso, Gilles; Bouvier, Jean-Louis; Comet, Bertrand
CORPORATE SOURCE: (1) Service de Cardiologie, Centre Hospitalier Prive Beauregard, 12 Impasse du Lido, 13012, Marseille France
SOURCE: Journal of the American College of Cardiology, (May, 2000) Vol. 35, No. 6, pp. 1543-1550. print.
ISSN: 0735-1097.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB OBJECTIVES: We sought to evaluate immediate and late outcomes after stenting for left main coronary artery (LMCA) stenosis. BACKGROUND: Conventional percutaneous transluminal coronary angioplasty (PTCA), for which coronary artery bypass grafting (CABG) has been the gold standard therapy for years, has yielded poor results in unprotected LMCA lesions. The development of coronary stents, together with their dramatic patency improvement provided by new antiplatelet regimens and their validation against restenosis, warrants a reappraisal of angioplasty in LMCA stenosis. METHODS: From January 1993 to September 1998, 140 consecutive unselected patients with unprotected LMCA stenosis underwent elective stenting. Group I included 47 high-CABG-risk patients, and group II included 93 low-CABG-risk patients. Ticlopidine without **aspirin** was routinely started at least 72 h before the procedure and continued for one month. Patients were reevaluated monthly. A follow-up angiography was requested after six months. RESULTS: The procedure success rate was 100%. One-month mortality was 9% (4/47) in group I and 0% in group II. A follow-up angiography was obtained in 82% of cases, and target lesion revascularization was required in 17.4%. One-year actuarial survival was 89% in the first 29 group I patients and 97.5% in the first 63 group II patients. CONCLUSIONS: Stenting of unprotected LMCA stenosis provided excellent immediate results, particularly in good CABG candidates. Medium-term results were good, with a restenosis rate of 23%, similar to that seen after stenting at other coronary sites. Stenting deserves to be considered a safe and effective alternative to CABG in institutions performing large numbers of PTCAs.

L9 ANSWER 3 OF 34 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000188421 MEDLINE
DOCUMENT NUMBER: 20188421 PubMed ID: 10723454
TITLE: [Interventional catheter treatment of bypass graft stenosis: comparison of intracoronary stent implantation and balloon angioplasty]. Katheterinterventionelle Therapie stenosierter Bypassgefasse. Vergleich zwischen intrakoronarer Stent-Implantation und Ballonangioplastie.
AUTHOR: Heidland U E; Heintzen M P; Schoppmann D; Michel C J; Strauer B E
CORPORATE SOURCE: Medizinische Klinik und Polyklinik B, Heinrich-Heine-Universitat Dusseldorf.. Heidland@med.uni-duesseldorf.de
SOURCE: DEUTSCHE MEDIZINISCHE WOCHENSCHRIFT, (2000 Feb 25) 125 (8) 206-10.
Journal code: ECL; 0006723. ISSN: 0012-0472.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000413
Last Updated on STN: 20000413
Entered Medline: 20000407
AB BACKGROUND AND OBJECTIVE: Balloon angioplasty of a stenosed bypass graft has a much higher risk of recurrent stenosis than dilatation of a stenosed

native coronary artery. Intracoronary stent implantation has established itself as the better treatment of native **coronary artery stenosis** than conventional balloon angioplasty. However, there is still uncertainty whether intracoronary stent implantation in stenosed bypass vessels gives better long-term results than conventional balloon angioplasty. PATIENTS AND METHODS: Results were retrospectively analyzed of unrandomized 224 primarily successful interventions--122 balloon dilatations and 102 stent implantations--performed between January 1996 and June 1998 on stenosed coronary bypass grafts, re-examined by coronary angiography an average of 6 months later. All but 11 patients were on combined **aspirin** and ticlopidine antiplatelet aggregation treatment. RESULTS: There was a significantly lower 6-month restenosis rate (30.4%) after stent implantation than after balloon dilatation (51.6%). The re-intervention rate was also significantly lower after stent implantation. CONCLUSION: These data suggest that stent implantation of stenosed coronary bypass grafts under cover of platelet-aggregation inhibition with **aspirin** and ticlopidine provides a lower restenosis and thus higher revascularization rate than conventional balloon dilatation.

L9 ANSWER 4 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2001:112474 BIOSIS
DOCUMENT NUMBER: PREV200100112474
TITLE: Intravascular beta-radiation with 32P reduces neointima after stent injury in porcine peripheral arteries.
AUTHOR(S): Kaluza, Grzegorz L. (1); Raizner, Albert E.; Schulz, Daryl G.; Tio, Fermin O.; Ali, Nadir M.
CORPORATE SOURCE: (1) Baylor Coll of Medicine, Houston, TX USA
SOURCE: Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.424. print.
Meeting Info.: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L9 ANSWER 5 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2001:105868 BIOSIS
DOCUMENT NUMBER: PREV200100105868
TITLE: Increased platelet activity after coronary stent implantation significantly correlates with high Lp(a) levels.
AUTHOR(S): Oemrawsingh, Pranobe V. (1); Hollaar, Leny (1); Jukema, Wouter J. (1); Nieuwland, Rienk (1); Van der Laarse, A. (1); Sturk, Auguste (1); Schalij, Martin J. (1)
CORPORATE SOURCE: (1) Leiden Univ Medical Ctr, Leiden Netherlands
SOURCE: Circulation, (October 31, 2000) Vol. 102, No. 18 Supplement, pp. II.334. print.
Meeting Info.: Abstracts from Scientific Sessions 2000 New Orleans, Louisiana, USA November 12-15, 2000
ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L9 ANSWER 6 OF 34 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998430436 MEDLINE
DOCUMENT NUMBER: 98430436 PubMed ID: 9759636
TITLE: The antiaggregating and antithrombotic activity of clopidogrel is potentiated by **aspirin** in several experimental models in the rabbit.

AUTHOR: Herbert J M; Dol F; Bernat A; Falotico R; Lale A; Savi P
CORPORATE SOURCE: Sanofi Recherche, Haemobiology Research Department, Toulouse, France.. jean-marc.herbert@tls1.elfsanofi.fr
SOURCE: THROMBOSIS AND HAEMOSTASIS, (1998 Sep) 80 (3) 512-8. Journal code: VQ7; 7608063. ISSN: 0340-6245.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981228

AB It is unknown whether the addition of **aspirin** might increase both the efficacy and the potency of clopidogrel, a potent and selective ADP blocker. For that purpose, the efficacy of clopidogrel (1-20 mg/kg, p.o.) administered orally to rabbits alone or in combination with **aspirin** (0.1-10 mg/kg, p.o.) was determined in several experimental models. A potent synergistic effect of the clopidogrel/ **aspirin** association was demonstrated with regard to collagen-induced platelet aggregation measured ex vivo. Similarly, **aspirin** potentiated the antithrombotic activity of clopidogrel measured with regard to experimental thrombosis induced by a silk thread or on stents placed in an arteriovenous shunt, thrombus formation following electrical stimulation of the rabbit carotid artery and with regard to 111In-labeled platelet deposition on a stent implanted in an arteriovenous shunt or on the subendothelium following air drying injury of the rabbit carotid artery. A similar potentiating effect of **aspirin** was obtained with regard to myointimal proliferation (restenosis) in the femoral arteries of atherosclerotic rabbits which occurred as a consequence of stent placement. The clopidogrel/ **aspirin** combination showed only additive-type effects on bleeding time prolongation induced by ear transection in the rabbit, therefore showing that combined inhibition of cyclooxygenase and ADP's effects provide a marked enhanced antithrombotic efficacy. Such a combination may provide substantial protection against platelet aggregation leading to thrombotic occlusion at sites of endothelial injuries and **coronary artery stenosis** in humans.

L9 ANSWER 7 OF 34 MEDLINE
ACCESSION NUMBER: 1998202860 MEDLINE
DOCUMENT NUMBER: 98202860 PubMed ID: 9541758
TITLE: Current knowledge and significance of coronary artery ectasia: a chronologic review of the literature, recommendations for treatment, possible etiologies, and future considerations.
AUTHOR: Sorrell V L; Davis M J; Bove A A
CORPORATE SOURCE: Department of Internal Medicine, East Carolina University School of Medicine, Greenville, North Carolina 27858, USA.
SOURCE: CLINICAL CARDIOLOGY, (1998 Mar) 21 (3) 157-60. Ref: 22
Journal code: DE9; 7903272. ISSN: 0160-9289.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980529
Last Updated on STN: 19980529
Entered Medline: 19980521

AB Coronary artery ectasia is the abnormal enlargement of the coronary

artery. The prognosis, treatment, and etiology of this disease remain an enigma. There is some evidence to suggest that the incidence of ectasia is increasing, and therefore understanding of this entity needs to improve. This article reviews the current literature on coronary artery ectasia and summarizes the findings. A treatment plan that targets each of the suggested clinical complications is provided. Using multiple indirect observations and current understanding of endothelium-derived relaxation factor, a possible etiology that implicates overstimulation of endogenous nitric oxide is provided. Current literature suggests that ectatic coronary arteries, even without the presence of coronary stenosis, are subject to thrombus formation, vasospasm, and spontaneous dissection. Newer subgroups of ectasia are arising with the use of multiple interventional devices to dilate **coronary artery stenosis**. By design, these destroy the media of the coronary artery, and it is not clear whether these "iatrogenic" ectatic arteries are subject to the same complications as "idiopathic" coronary artery ectasia. Further investigation is necessary to help define the benefit of the proposed treatment regimen, to clarify the prognosis of these newer groups of "iatrogenic" ectasia, and to confirm or disprove the hypothesis targeting nitric oxide as an etiologic factor.

L9 ANSWER 8 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1998:89456 BIOSIS
DOCUMENT NUMBER: PREV199800089456
TITLE: Stenting of unprotected left main coronary artery stenoses: Immediate and late outcomes.
AUTHOR(S): Park, Seung-Jung (1); Park, Seong-Wook; Hong, Myeong-Ki; Cheong, Sang-Sig; Lee, Cheol Whan; Kim, Jae-Joong; Hong, Mun K.; Mintz, Gary S.; Leon, Martin B.
CORPORATE SOURCE: (1) Dep. Intern. Med., Coll. Med., Univ. Ulsan, Cardiovasc. Cent., Asan Med. Cent., 388-1 Pungnap-dong, Songpa-gu, Seoul South Korea
SOURCE: Journal of the American College of Cardiology, (Jan., 1998) Vol. 31, No. 1, pp. 37-42.
ISSN: 0735-1097.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Objectives. We examined the immediate and long-term out. comes after stenting of unprotected left main coronary artery (LMCA) stenoses in patients with normal left ventricular (LV) function. Background. Left main coronary artery disease is regarded as an absolute contraindication for coronary angioplasty. Recently, several reports on protected or unprotected LMCA stenting, or both, suggested the possibility of percutaneous intervention for this prohibited area. Methods. Forty-two consecutive patients with unprotected LMCA stenoses and normal LV function were treated with stents. The post-stent antithrombotic regimens were **aspirin** and ticlopidine; 14 patients also received warfarin. Patients were followed very closely with monthly telephone interviews and follow-up angiography at 6 months. Results. The procedural success rate was 100%, with no episodes of subacute thrombosis regardless of anticoagulation regimen. Six-month follow-up angiography was performed in 32 of 34 eligible patients. Angiographic restenosis occurred in seven patients (22%, 95% confidence interval 7% to 37%); five patients subsequently underwent elective coronary artery bypass graft surgery (CABG), and two patients were treated with rotational atherectomy plus adjunct balloon angioplasty. The only death occurred 2 days after elective CABG for treatment of in-stent restenosis. The other patients (without angiographic follow-up) remain asymptomatic. Conclusions. Stenting of unprotected LMCA stenoses may be a safe and effective alternative to CABG in carefully selected patients with normal LV function. Further studies in larger patient populations are needed to assess late outcome.

L9 ANSWER 9 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1997:505730 BIOSIS
DOCUMENT NUMBER: PREV199799804933
TITLE: Comparison of enoxaparin, hirulog, and heparin as adjunctive antithrombotic therapy during thrombolysis with rtPA in the stenosed canine coronary artery.
AUTHOR(S): Leadley, Robert J., Jr. (1); Kasiewski, Charles J.; Bostwick, Jeffrey S.; Bentley, Ross; McVey, Matthew J.; White, Francis J.; Perrone, Mark H.; Dunwiddie, Christopher T.
CORPORATE SOURCE: (1) Cardiovasc. Biol., Mail Stop: NW4, Rhone-Poulenc Rorer Central Res., 500 Arcola Rd., Collegeville, PA 19426 USA
SOURCE: Thrombosis and Haemostasis, (1997) Vol. 78, No. 4, pp. 1278-1285.
ISSN: 0340-6245.
DOCUMENT TYPE: Article
LANGUAGE: English
AB A canine model of electrolytic injury-induced coronary artery thrombosis and rtPA-induced thrombolysis was used to evaluate the relative antithrombotic efficacy of enoxaparin (a low molecular weight heparin), conventional therapy (heparin or heparin plus **aspirin**), and hirulog (a direct thrombin inhibitor), when used as adjunctive therapy during thrombolysis. After 60 min of clot aging, adjunctive therapy was begun at doses which elevated APTT approximately 2-fold over baseline. Fifteen minutes after the start of adjunctive therapy, recombinant tissue plasminogen activator (rtPA) was administered (100 mu-g/kg i.v. bolus + 20 mu-g/kg/min for 60 min). Adjunctive therapy continued for 1 h after termination of rtPA and blood flow was monitored for two additional hours. Enoxaparin (1 mg/kg i.v. bolus + 30 mu-g/kg/min, n = 10 for each treatment group) was the only adjunctive treatment that significantly increased the total minutes of flow (143 +- 25 min out of a possible 240 min, vs 54 +- 25 min for vehicle, p lt 0.05) and decreased thrombus mass (6.0 +- 1.3 mg vs 11.8 +- 3.2 mg for vehicle). Although hirulog (2 mg/kg i.v. bolus + 40 mu-g/kg/min) did not significantly increase the minutes of flow (120 +- 27 min, p lt 0.06) or decrease thrombus mass (8.7 +- 1.7 mg) compared to vehicle, these values were not significantly different than those measured in the enoxaparin group. However, the results with hirulog were achieved at the expense of a significantly greater increase in template bleeding time than that measured during enoxaparin treatment. Minutes of flow for heparin (50 U/kg i.v. bolus + 0.6 U/kg/min) and heparin plus **aspirin** (5 mg/kg i.v. bolus) were 69 +- 20 and 60 +- 23 min, respectively; thrombus masses were 8.2 +- 1.3 and 7.3 +- 1.0 mg, respectively. In summary, enoxaparin was more effective than conventional therapy in this model in terms of vessel patency and thrombus mass, and was as effective as hirulog, at least at a dose of hirulog that only modestly impaired hemostasis. Therefore, enoxaparin may prove to be a safe and effective alternative agent for adjunctive therapy during thrombolysis with rtPA.

L9 ANSWER 10 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1997:538547 BIOSIS
DOCUMENT NUMBER: PREV199799837750
TITLE: Factors contributing to the progression rate of **coronary artery stenosis**.
AUTHOR(S): Sunagawa, Osahiko; Touma, Takashi; Imai, Chiharu; Uechi, Yoichi; Kamiyama, Tomomasa; Okumura, Koichiro; Ishikawa, Naoki; Hata, Yoshio; Wakugami, Kiyoshi; Kimura, Yorio; Fukiyama, Koshiro
CORPORATE SOURCE: Third Dep. Internal Med., University Ryukyus Sch. Med., Tokyo Japan
SOURCE: Japanese Circulation Journal, (1997) Vol. 61, No. 7, pp. 632.

Meeting Info.: 61st Annual Scientific Meeting of the Japanese Circulation Society Tokyo, Japan March 31-April 2, 1997
ISSN: 0047-1828.
DOCUMENT TYPE: Conference; Abstract; Conference
LANGUAGE: English

L9 ANSWER 11 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1997:480414 BIOSIS
DOCUMENT NUMBER: PREV199799779617
TITLE: Adenoviral transfer of human constitutive endothelial nitric oxide synthase gene inhibits restenosis after angioplasty in porcine coronary arteries.
AUTHOR(S): Varenne, O. (1); Gillijns, H. (1); Sinnaeve, P.; Pislaru, S.; Van Pelt, N. (1); Vermeersch, P. (1); Gerard, R. (1); Collen, D. (1); Van De Werf, F.; Janssens, S.
CORPORATE SOURCE: (1) Cent. Transgene Technol. Gene Ther., Leuven Belgium
SOURCE: European Heart Journal, (1997) Vol. 18, No. ABSTR. SUPPL., pp. 459.
Meeting Info.: XIXth Congress of the European Society of Cardiology together with the 32nd Annual General Meeting of the Association of European Paediatric Cardiologists (AEPC) Stockholm, Sweden August 24-28, 1997
ISSN: 0195-668X.
DOCUMENT TYPE: Conference; Abstract
LANGUAGE: English

L9 ANSWER 12 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1997:185508 BIOSIS
DOCUMENT NUMBER: PREV199799484711
TITLE: Gastric administration of a commercial flavonoid inhibits in vivo and ex vivo platelet aggregation in dogs with stenosed coronary arteries.
AUTHOR(S): Osman, H. E.; Maalej, N.; Folts, J. D.
CORPORATE SOURCE: Univ. Wisconsin-Madison, Madison, WI 53792 USA
SOURCE: FASEB Journal, (1997) Vol. 11, No. 3, pp. A314.
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology 97 New Orleans, Louisiana, USA April 6-9, 1997
ISSN: 0892-6638.
DOCUMENT TYPE: Conference; Abstract
LANGUAGE: English

L9 ANSWER 13 OF 34 MEDLINE
ACCESSION NUMBER: 97297195 MEDLINE
DOCUMENT NUMBER: 97297195 PubMed ID: 9152664
TITLE: Treatment of ischaemic heart disease. Role of drugs, surgery and angioplasty in unstable angina patients.
AUTHOR: Conti C R
CORPORATE SOURCE: Department of Medicine and Cardiology, University of Florida, College of Medicine, Gainesville 32610-0277, USA.
SOURCE: EUROPEAN HEART JOURNAL, (1997 May) 18 Suppl B B11-5. Ref: 25
Journal code: EM8; 8006263. ISSN: 0195-668X.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970812

Last Updated on STN: 19970812
Entered Medline: 19970731

AB The term unstable angina should only be used to describe patients whose immediate prognosis is uncertain and the nature of the unstable disease may vary on a patient to patient basis, making broad categorization of such patients inappropriate. Unstable angina may be caused by extracardiac factors, such as uncontrolled hypertension and tachycardia, disruption of an atheromatous plaque, dynamic or intermittent coronary artery thrombosis, haemorrhagic dissection into an atheromatous plaque, epicardial coronary spasm or progression of atherosclerosis as a result of plaque healing. Control of symptoms using medical therapy with a combination of nitrates, beta-blockers and calcium antagonists is usually quite successful. In the absence of contra-indications, intravenous heparin, and possibly anti-platelet agents, should also be used in the acute phase of treatment. In addition, one **aspirin** a day is indicated unless there are definite contra-indications. If symptoms are relieved, evaluation and management should proceed as with chronic stable angina. Identification of patients with a poor prognosis should be the main indication for urgent revascularization. One of the best predictors of a poor prognosis in unstable disease is persistent pain despite optimum therapy. Urgent surgery should be considered in any patient with multivessel **coronary artery stenosis** who has evidence of persistent myocardial ischaemia, despite adequate medical therapy.

L9 ANSWER 14 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1996:146818 BIOSIS
DOCUMENT NUMBER: PREV199698718953
TITLE: Stenting of unprotected left main **coronary artery stenosis** without coumadin.
AUTHOR(S): Fajadet, Jean; Brunel, Philippe; Jordan, Christian; Cassagneau, Bernard; Marco, Jean
CORPORATE SOURCE: Clinique Pasteur, Toulouse France
SOURCE: Journal of the American College of Cardiology, (1996) Vol. 27, No. 2 SUPPL. A, pp. 277A.
Meeting Info.: 45th Annual Scientific Session of the American College of Cardiology Orlando, Florida, USA March 24-27, 1996
ISSN: 0735-1097.
DOCUMENT TYPE: Conference
LANGUAGE: English

L9 ANSWER 15 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1996:560261 BIOSIS
DOCUMENT NUMBER: PREV199699282617
TITLE: Elective stenting for the treatment of lesions located in small coronary arteries.
AUTHOR(S): Romero, M. (1); Suarez De Lezo, J. (1); Medina, A.; Pan, M.; Hernandez, E.; Segura, J.; Melian, F.; Ruiz, M.; Zayas, R.; Ortega, J. R.
CORPORATE SOURCE: (1) Univ. Cordoba, Cordoba Spain
SOURCE: European Heart Journal, (1996) Vol. 17, No. ABSTR. SUPPL., pp. 217.
Meeting Info.: XVIIIth Congress of the European Society of Cardiology Birmingham, England, UK August 25-29, 1996
ISSN: 0195-668X.
DOCUMENT TYPE: Conference
LANGUAGE: English

L9 ANSWER 16 OF 34 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96368088 MEDLINE
DOCUMENT NUMBER: 96368088 PubMed ID: 8772241

TITLE: Inhibition of thrombus formation by endothelin-1 in canine models of arterial thrombosis.
AUTHOR: Leadley R J Jr; Humphrey W R; Erickson L A; Shebuski R J
CORPORATE SOURCE: Cardiovascular Diseases Research, Upjohn Laboratories, Kalamazoo, MI, USA.
SOURCE: THROMBOSIS AND HAEMOSTASIS, (1995 Dec) 74 (6) 1583-90.
Journal code: VQ7; 7608063. ISSN: 0340-6245.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 19961106
Last Updated on STN: 19961106
Entered Medline: 19961023

AB The effect of endothelin-1 (ET-1) on thrombus formation in vivo was evaluated in two well-established canine models of coronary artery thrombosis. First, the possible antithrombotic effect of ET-1 was examined using the cyclic flow reduction (CFR) model of **coronary artery stenosis**, vascular endothelial cell and intimal smooth muscle cell injury, and periodic acute platelet thrombus formation. Using a rating system of 0 (no inhibition) to 3 (complete inhibition), ET-1 administration at 0.1, 0.5, and 1.0 microgram/kg, i.v. bolus, produced scores of 1.0 +/- 0.2 (n = 10), 1.8 +/- 0.4 (n = 8), and 2.1 +/- 0.3 (n = 7), respectively. ET-1 injection inhibited ex vivo platelet aggregation induced by ADP and U-46619 by 30-60%. When **aspirin** was administered at 5 mg/kg prior to ET-1 administration at 0.5 microgramoff, ET-1 produced a CFR rating of 2.7 +/- 0.2 (n = 6). However, higher dose **aspirin** (30 mg/kg, i.v.) significantly inhibited the antithrombotic effect of ET-1 (0.5 +/- 0.5, n = 4). The antithrombotic effect of ET-1 was also examined using an electrolytic injury model of arterial thrombosis. The time required to produce an occlusive thrombus during the experiments in which ET-1 was administered at 10 and 20 ng.kg-1.min-1 was 77 +/- 15 ($p < 0.08$) and 105 +/- 16 min ($p < 0.05$), respectively, compared to 44 +/- 5 min when vehicle was infused. Cardiovascular changes following occlusion were not significantly different between dogs given ET-1 and those given vehicle, suggesting that elevated plasma levels of ET-1 did not exacerbate the adverse effects of coronary occlusion. In addition, plasma ET-1 levels were elevated significantly after occlusion in the dogs given vehicle (from 7.4 to 12.4 pg/ml). Taken together, these date provide further evidence to support the notion that ET-1 release during ischemia may be involved in a protective mechanism that impeded thrombus formation in the stenosed coronary artery.

L9 ANSWER 17 OF 34 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96100197 MEDLINE
DOCUMENT NUMBER: 96100197 PubMed ID: 8569218
TITLE: Failure of calcium channel blockade to reduce platelet-mediated cyclic flow variations in dogs with coronary stenosis and endothelial injury.
AUTHOR: Beaughard M; Brasset M; John G; Massingham R
CORPORATE SOURCE: RL-CERM Riom, France.
SOURCE: JOURNAL OF CARDIOVASCULAR PHARMACOLOGY, (1995 Oct) 26 (4) 577-83.
Journal code: K78; 7902492. ISSN: 0160-2446.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199603
ENTRY DATE: Entered STN: 19960315
Last Updated on STN: 19960315

Entered Medline: 19960307

AB Experimental canine **coronary artery stenosis** associated with endothelial injury results in a typical pattern of coronary flow characterized by gradual decreases in blood flow to almost zero values followed by abrupt restorations to original levels. Cyclic flow variations (CFVs) are the consequence of recurrent platelet aggregation at the site of the stenosis and subsequent dislodgement of the thrombus. The present study was designed to test the efficacy of diltiazem, nifedipine, and verapamil in inhibiting in vivo platelet aggregation as compared with that of **aspirin** and ketanserin, two potent reference compounds effective in this model. Except for **aspirin**, compounds were given as a slow intravenous infusion (i.v.) for 60 min to avoid hemodynamic changes. Diltiazem (0.01 mg/kg/min), nifedipine (3 micrograms/kg/min), and verapamil (0.01 mg/kg/min) were totally inactive against CFVs. A higher dose of verapamil (0.02 mg/kg/min) abolished CFVs in 3 of 4 dogs, but serious side effects were observed [atrioventricular (AV) block and death of 2 animals]. **Aspirin** (10 mg/kg bolus) caused complete inhibition of CFVs in 4 of 4 dogs, and ketanserin (0.01 mg/kg/min) abolished CFVs in 4 of 5 dogs. These data suggest that calcium channel blockade alone in contrast to cyclooxygenase inhibition or 5-HT2 antagonism cannot inhibit thrombus formation in this model.

L9 ANSWER 18 OF 34 MEDLINE DUPLICATE 5

ACCESSION NUMBER: 95096385 MEDLINE
DOCUMENT NUMBER: 95096385 PubMed ID: 7798505
TITLE: Frequent reocclusion of patent infarct-related arteries between 4 weeks and 1 year: effects of antiplatelet therapy.
AUTHOR: White H D; French J K; Hamer A W; Brown M A; Williams B F; Ormiston J A; Cross D B
CORPORATE SOURCE: Cardiology Department, Green Lane Hospital, Auckland, New Zealand.
SOURCE: JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY, (1995 Jan) 25 (1) 218-23.
Journal code: H50; 8301365. ISSN: 0735-1097.
PUB. COUNTRY: United States
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199501
ENTRY DATE: Entered STN: 19950215
Last Updated on STN: 19960129
Entered Medline: 19950125

AB OBJECTIVES. This study assessed the effect of the combination of **aspirin** and dipyridamole on patency of the infarct-related artery between 4 weeks and 1 year after myocardial infarction. BACKGROUND. Patency of the infarct-related artery is an important determinant of prognosis after myocardial infarction. The incidence of late reocclusion and the effects of antiplatelet therapy are unknown. METHODS. To investigate the importance of antiplatelet therapy for the prevention of late reocclusion, 215 patients who had a patent infarct-related artery 4 weeks after myocardial infarction were randomized in a double-blind manner to receive either a combination of 25 mg of **aspirin** and 200 mg of dipyridamole twice daily or placebo. One hundred fifty-four patients underwent further coronary arteriography 1 year later. RESULTS. At 1 year, 38 (25%) of 154 patients had reocclusion of the infarct-related artery; 18 (23%) of 79 patients receiving **aspirin** and dipyridamole had late reocclusion versus 20 (27%) of 75 who received placebo (p = NS). The rate of reocclusion was related to the severity of the residual

coronary artery stenosis at 4 weeks (< 50% stenosis 9.2%; 50% to 69% stenosis 11.6%; 70% to 89% stenosis 30.4%; > or = 90% stenosis 70%, p < 0.01). The majority of reocclusions were silent, and only 17 (45%) of 38 were clinically associated with further infarction. There were no differences for a hierarchic end point of cardiac death, myocardial infarction or revascularization (14.8% **aspirin** and dipyridamole vs. 17.8% placebo). CONCLUSIONS. Late reocclusion of the patent infarct-related artery is a frequent event, occurring in 25% of patients. Antiplatelet therapy with the combination of **aspirin** and dipyridamole does not alter the overall rate of late reocclusion. Other strategies are required to reduce late reocclusion.

L9 ANSWER 19 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1995:256939 BIOSIS
DOCUMENT NUMBER: PREV199598271239
TITLE: Drugs for the prevention of coronary thrombosis: From an animal model to clinical trials.
AUTHOR(S): Folts, John David
CORPORATE SOURCE: Cardiol. Sect., H6/379, Univ. Wis., Clin. Sci. Cent., 600 Highland Ave., Madison, WI 53792-3248 USA
SOURCE: Cardiovascular Drugs and Therapy, (1995) Vol. 9, No. SUPPL. 1, pp. 31-43.
ISSN: 0920-3206.
DOCUMENT TYPE: General Review
LANGUAGE: English
AB Platelets contribute to the progression of atherosclerotic disease and also to partial or complete thrombotic occlusion of stenosed human coronary or cerebral arteries. Thus, there is considerable interest in being able to measure in vivo or ex vivo platelet function or level of activity. Currently, platelet activity and the platelet inhibitory effect of drugs can be assessed ex vivo or in vitro by platelet aggregometry. There is also an experimental animal model (the cyclic flow, or Folts, model) for studying the interactions of platelets with damaged and stenosed arterial walls. This model was first used to show that **aspirin** can prevent coronary thrombosis in stenosed canine coronary arteries and is fairly predictive in determining which drugs are likely to inhibit platelet activity in vivo. It is also useful in identifying which drugs may be beneficial in ameliorating unstable angina and preventing coronary thrombosis. Studies with this model predict that **aspirin**, sulfinpyrazone, the monoclonal antibody 7E3 to the platelet glycoprotein GpIIb-IIIa fibrinogen receptor, arginine-glycine-aspartic acid peptide mimetics, or clopidogrel (an analogue of ticlopidine) would inhibit platelet-mediated thrombosis in patients with coronary or cerebral artery stenosis. The model also predicts that heparin or dipyridamole alone would not prevent platelet-mediated arterial thrombosis. Finally, studies with the cyclic model suggest that while serotonin receptor blockers, alpha-adrenergic blockade, or infusions of prostacyclin (or its analogue, Iloprost) would inhibit platelet activity, the resulting hypotension would severely limit the clinical usefulness of these compounds.

L9 ANSWER 20 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1994:454053 BIOSIS
DOCUMENT NUMBER: PREV199497467053
TITLE: Thromboxane receptor antagonist BMS-180291, but not **aspirin**, reduces the severity of pacing-induced ischemia in dogs.
AUTHOR(S): Grover, Gary J. (1); Schumacher, William A.; Ogletree, Martin L.
CORPORATE SOURCE: (1) Dep. Pharmacool., Bristol-Myers Squibb Pharm Res. Inst., P.O. Box 4000, Princeton, NJ 08543-4000 USA
SOURCE: Journal of Cardiovascular Pharmacology, (1994) Vol. 24, No.

3, pp. 493-499.
ISSN: 0160-2446.
DOCUMENT TYPE: Article
LANGUAGE: English
AB We determined the effect of thromboxane A-2 (TXA-2) prostaglandin endoperoxide (TP) receptor antagonism, using BMS-180291 or **aspirin**, on the severity of pacing-induced ischemia in anesthetized dogs. Thromboxane receptor antagonists may not only have antithrombotic activity, but may also have direct cardioprotective effects, unlike **aspirin**. Left anterior descending coronary artery (LAD) stenosis was adjusted so that a significant (10-12 mV) ST segment elevation was observed only when superimposed on atrial pacing. Each heart was subjected to 5-min episodes of pacing-induced ischemia 10, 40, and 70 min after initiation of BMS-180291 (1 mg/kg + 1 mg/kg/h) or vehicle. In the vehicle group, ST segment elevation was reproducible at all pacing-induced ischemia episodes, whereas BMS-180291 significantly reduced it by 30% at the later ischemia episodes. This reduction in ST segment increase was not accompanied by alterations in regional myocardial blood flow (RMBF) nor in hemodynamic status. **Aspirin** in the same model (10 mg/kg intravenously (i.v.) given 10 min before pacing-induced ischemial did not significantly reduce ST segment elevation, indicating a lack of protective effect in this model. Thromboxane receptor blockade appears to protect myocardium subjected to pacing-induced ischemia, an effect not produced by **aspirin**.

L9 ANSWER 21 OF 34 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 93249903 MEDLINE
DOCUMENT NUMBER: 93249903 PubMed ID: 8485070
TITLE: Effects of trimetazidine on in vivo coronary arterial platelet thrombosis.
AUTHOR: Belcher P R; Drake-Holland A J; Hynd J W; Noble M I
CORPORATE SOURCE: Academic Unit of Cardiovascular Medicine, Charing Cross and Westminster Medical School, London, England.
SOURCE: CARDIOVASCULAR DRUGS AND THERAPY, (1993 Feb) 7 (1) 149-57.
Journal code: AYG; 8712220. ISSN: 0920-3206.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199306
ENTRY DATE: Entered STN: 19930618
Last Updated on STN: 19930618
Entered Medline: 19930608
AB We used Folts' model of critical **coronary artery stenosis** with endothelial damage, which measures platelet-rich thrombus accumulation from cyclic flow reductions (CFRs). This paper reports results applied to trimetazidine, a member of the piperazine group. Trimetazidine at a dose of 1 mg/kg completely abolished CFRs caused by accumulating thrombus in the circumflex coronary artery in 4 of 8 open-chest anesthetized beagles. More trimetazidine (up to 5 mg/kg) abolished CFRs in two more and attenuated them in the remaining two dogs. There were no systemic hemodynamic effects observed. Adrenaline was then infused to stimulate platelet activation. At a rate of 0.4 microgram/kg/min, CFRs were restored in one dog only. Adrenaline given at 1.6 micrograms/kg/min resulted in restoration or increase in the slope of CFRs in all animals. A further six nonoperated dogs were anesthetized and given trimetazidine 3 mg/kg. Routine coagulation studies were not altered. However, **aspirin** 5 mg/kg significantly increased bleeding time, whereas trimetazidine alone did not. These findings suggest that trimetazidine is effective in preventing intracoronary platelet aggregation in this model. Because of its demonstrated sparing of coagulation factors and its lack of effect on bleeding time, the cause is

unlikely to be inhibition of the fibrinogen or thrombin receptors, or interference with arachidonic acid metabolism.

L9 ANSWER 22 OF 34 MEDLINE
ACCESSION NUMBER: 93099577 MEDLINE
DOCUMENT NUMBER: 93099577 PubMed ID: 8416335
TITLE: Platelet adhesion/aggregation in an in vitro model of **coronary artery stenosis.**
AUTHOR: Grabowski E F; Rodriguez M; McDonnell S L
CORPORATE SOURCE: Department of Pediatrics, New York Hospital-Cornell Medical Center, New York.
CONTRACT NUMBER: HL 33095 (NHLBI)
SOURCE: CATHETERIZATION AND CARDIOVASCULAR DIAGNOSIS, (1993 Jan) 28 (1) 65-71.
Journal code: CQZ; 7508512. ISSN: 0098-6569.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199301
ENTRY DATE: Entered STN: 19930205
Last Updated on STN: 19930205
Entered Medline: 19930121

AB Platelet adhesion/aggregation (PAA) at a site of **coronary artery stenosis** is believed to be a process strongly modulated by local shear rates and the functional state of neighboring endothelium. One purpose of the present work, therefore, is to describe an in vitro model for the direct imaging of such PAA. Another is to apply the model to the question as to whether the use of nonionic vs. ionic contrast media (CM) in the presence of vascular endothelium contributes to PAA at the stenosis site. Toward these ends, we utilized a special flow chamber which incorporates a monolayer of endothelial cells (ECs), a step 66% flowpath constriction at a site preadsorbed with microfibrillar collagen, and arterial shear rates. By epifluorescence microscopy and digital image analysis of video recordings, PAA was found to be greater with dysfunctional ECs (pretreated with lysine acetylsalicyclate) than with normal ECs, thereby confirming a modulatory role in PAA of functionally intact ECs. When nonionic (iohexol) or ionic (ioxaglate, diatrizoate) CM was added to the flowing blood at a concentration of 20% by non-red cell volume, PAA was inhibited in the order diatrizoate > ioxaglate > iohexol > saline control. No inhibition by any CM was seen, however, when chamber prefill culture medium containing 20% by volume CM was displaced by CM-free blood, in simulation of bolus administration of CM. In terms of inhibition of PAA during percutaneous transluminal coronary angioplasty (PTCA), therefore, our model provides a conceptual basis by which one may anticipate in flowing blood no clear benefit of ionic over nonionic CM.(ABSTRACT TRUNCATED AT 250 WORDS)

L9 ANSWER 23 OF 34 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 92351357 MEDLINE
DOCUMENT NUMBER: 92351357 PubMed ID: 1641819
TITLE: Experimental carotid stenosis and endothelial injury in the rabbit: an in vivo model to study intravascular platelet aggregation.
AUTHOR: Golino P; Ambrosio G; Pascucci I; Ragni M; Russolillo E; Chiariello M
CORPORATE SOURCE: Division of Cardiology, 2nd School of Medicine, University of Naples, Italy.
SOURCE: THROMBOSIS AND HAEMOSTASIS, (1992 Mar 2) 67 (3) 302-5.
Journal code: VQ7; 7608063. ISSN: 0340-6245.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199208
ENTRY DATE: Entered STN: 19920911
Last Updated on STN: 19980206
Entered Medline: 19920828

AB Previous studies have shown that experimental canine **coronary artery stenosis** associated with endothelial injury results in a typical pattern of coronary flow characterized by gradual decreases in coronary flow to almost zero values followed by restorations of flow to normal values. This pattern of flow, called cyclic flow reductions (CFRs), is the consequence of recurrent platelet aggregation at the site of the stenosis and endothelial injury and subsequent dislodgement of the thrombus. In the present study, platelet activation and aggregation in vivo was induced by placing an external constrictor around carotid arteries with endothelial injury in anesthetized rabbits. Carotid blood flow velocity was measured continuously with a Doppler flow probe positioned proximally to the constrictor. After placement of the constrictor, CFRs developed in 14 of 14 rabbits with a mean frequency of 16.5 +/- 2.3 cycles/h. CFRs were observed for 30 min, and the animals were treated with either an i.v. bolus of **aspirin** (10 mg/kg) or R 68070 (20 mg/kg), a drug with simultaneous TxA2 synthase and TxA2/PGH2 receptor blocking properties. **Aspirin** completely inhibited CFRs in 4 of 7 rabbits, whereas R 68070 eliminated CFRs in 7 of 7 animals. In the 3 animals that did not respond to **aspirin**, administration of ketanserin (0.25 mg/kg i.v.), a selective serotonin S2 receptor antagonist, completely abolished CFRs. Both **aspirin** and R 68070 resulted in a marked reduction in serum TxB2 formation and in a complete inhibition of ex vivo platelet aggregation in response to arachidonic acid, whereas aggregation in response to U46619, a TxA2 mimetic, was inhibited only in R 68070-treated rabbits.(ABSTRACT TRUNCATED AT 250 WORDS)

L9 ANSWER 24 OF 34 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 92001947 MEDLINE
DOCUMENT NUMBER: 92001947 PubMed ID: 1911705
TITLE: Effect of **aspirin** and epinephrine on experimentally induced thrombogenesis in dogs. A parallelism between in vivo and ex vivo thrombosis models.
AUTHOR: Roux S P; Sakariassen K S; Turitto V T; Baumgartner H R
CORPORATE SOURCE: Pharma Division, Preclinical Research/PRPV, F. Hoffmann-La Roche Ltd., Basel, Switzerland.
SOURCE: ARTERIOSCLEROSIS AND THROMBOSIS, (1991 Sep-Oct) 11 (5) 1182-91.
Journal code: AZ1; 9101388. ISSN: 1049-8834.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199110
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19920124
Entered Medline: 19911025

AB Thrombosis on the damaged or ruptured vascular wall in a stenotic coronary artery is believed to be the precipitating factor leading to unstable angina. Little is known about the nature of the interactions among platelets, fluid dynamic factors, and vessel wall properties under such conditions. In the present investigation we have compared two experimental models of thrombosis simultaneously in anesthetized dogs. The first was an in vivo model of unstable angina, in which a fixed circumflex **coronary artery stenosis** was produced and the resultant cyclic blood flow reductions (CFRs) through the vessel were

investigated after infusion of **aspirin** and a combination of **aspirin** and epinephrine. As previously reported, **aspirin** inhibited the CFRs, but the continuous infusion of epinephrine reestablished the appearance of CFRs. The second was an ex vivo model, in which thrombus formation on a type III collagen surface was investigated in a parallel-plate perfusion system under controlled conditions of exposure time and flow; morphological evaluation of thrombus volume, platelet adhesion, and fibrin deposition was performed. The chamber was positioned in an extracorporeal shunt between the carotid artery and the jugular vein of anesthetized dogs and exposed to nonanticoagulated blood at a shear rate of 1,600 sec-1. Thirty minutes after establishment of the CFRs, a blood sample for platelet aggregation was collected and a bleeding time and a first ex vivo perfusion were performed. At the end of this perfusion, animals were subjected either to no treatment (n = 10) or to an intravenous bolus of 10 mg/kg **aspirin** (n = 7), and a second perfusion was conducted 30 minutes later. Additional untreated animals (n = 6) were given **aspirin** followed by a continuous intravenous infusion of 10 micrograms/ml epinephrine, and a third perfusion was conducted. Results with respect to platelet adhesion, thrombus volume, and fibrin deposition were similar in the two perfusions in untreated animals. Treatment with **aspirin** abolished the CFRs in all dogs and concomitantly reduced the ex vivo thrombus volume by 84% (p less than 0.01) without affecting platelet adhesion and fibrin deposition. Bleeding time increased by 40% (p less than 0.05), and collagen-induced platelet aggregation was virtually abolished (p less than 0.01). However, infusion of epinephrine in dogs after **aspirin** treatment restored the CFRs, and the ex vivo thrombus volumes were not statistically different from predrug values. Thus, the ex vivo model satisfactorily reflects the more complicated in vivo model events with respect to intracoronary thrombosis and substantiates the view that **aspirin** interrupts coronary thrombogenesis in the dog by interfering with platelet cohesion.

L9 ANSWER 25 OF 34 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 91176880 MEDLINE
DOCUMENT NUMBER: 91176880 PubMed ID: 2007377
TITLE: [Effects of high and low doses of acetylsalicylic acid on the restenosis rate after initially successful coronary angioplasty].
Wirkung hoher und niedriger Dosen Acetylsalicylsaure auf die Re-Stenosierungsrate nach primar erfolgreicher koronarer Angioplastie.
AUTHOR: Schanzenbacher P; Grimme M; Walter U; Kochsiek K
CORPORATE SOURCE: Medizinische Klinik, Universitat Wurzburg.
SOURCE: DEUTSCHE MEDIZINISCHE WOCHENSCHRIFT, (1991 Mar 29) 116 (13) 481-5.
Journal code: ECL; 0006723. ISSN: 0012-0472.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
(CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199105
ENTRY DATE: Entered STN: 19910519
Last Updated on STN: 19970203
Entered Medline: 19910502

AB A comparison was made in 79 patients (63 men, 16 women: mean age 52 +/- 9 years) of the effect of high and low doses of **aspirin** on restenosis rate during the first six months after originally successful percutaneous transluminal coronary angioplasty (PTCA), 39 patients (group 1) received 1000 mg **aspirin** daily, while 40 (group 2) received 100 mg daily. All patients took 1000 mg **aspirin** as loading dose

on the day before PTCA, and additionally calcium antagonists and slow-release nitrates in the post-PTCA period. Both groups were comparable with respect of localization of the dilated **coronary artery stenosis** and the morphological changes after dilatation. Intimal lesions after PTCA were demonstrated in 9 patients of group 1 and 10 of group 2. Within six months clinically significant restenosis had occurred in 8 patients of group 1 and 7 of group 2. 33 patients in group 2 and 31 in group 1 were free of symptoms and had no ischaemic reaction on the exercise ECG six months after the initial successful PTCA. These results demonstrate that high **aspirin** dosage does not reduce the restenosis rate more than low dosage.

L9 ANSWER 26 OF 34 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 92055478 MEDLINE
DOCUMENT NUMBER: 92055478 PubMed ID: 1948816
TITLE: Antithrombotic activity of BMY-43351, a new imidazoquinoline with enhanced aqueous solubility.
AUTHOR: Fleming J S; Buchanan J O; Seiler S M; Meanwell N A
CORPORATE SOURCE: Department of Cardiovascular Biochemistry, Bristol-Myers Squibb Institute for Pharmaceutical Research, Wallingford, CT 06492-7660.
SOURCE: THROMBOSIS RESEARCH, (1991 Jul 1) 63 (1) 145-55.
Journal code: VRN; 0326377. ISSN: 0049-3848.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199112
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19920124
Entered Medline: 19911219

AB BMY-43351 is a new broad-spectrum inhibitor of platelet aggregation with greater aqueous solubility than earlier analogs from the imidazoquinoline series. This report compares the antithrombotic activity of BMY-43351 to that of two other imidazoquinolines: BMY-20844, a simply-substituted compound, and BMY-21638, a more potent ether-linked side chain analog. All of these compounds act, at least in part, via inhibition of platelet low-Km cyclic AMP phosphodiesterase. Antithrombotic activity was assessed in the rabbit ear chamber-biolaser preparation, an animal model of small vessel thrombosis, and in the canine **coronary artery stenosis**-occlusion model of large vessel thrombosis. BMY-43351 was found to be remarkably potent in the biolaser model, with an EDso of 0.074 mg/kg p.o. In comparison, compounds such as **aspirin**, ticlopidine, sulfinpyrazone, and dipyridamole demonstrate little or no activity at much higher doses, (eg. 100 mg/kg p.o.). Other inhibitors of platelet low Km cyclic AMP phosphodiesterase are active but substantially weaker than BMY-43351. Similarly, in the **coronary artery stenosis**-occlusion model, BMY-43351 demonstrated impressive activity, significantly inhibiting arterial thrombosis at intraduodenal doses as low as 1 micrograms/kg. The potential use of BMY-43351 as adjunct therapy in thrombolysis was suggested in a series of experiments where this drug was used in combination with a thrombolytic regimen of stretokinase plus heparin. In this experimental setting, time to reperfusion was reduced from 42 +/- 5 minutes to 11 +/- 5 minutes, and reocclusion was totally inhibited.

L9 ANSWER 27 OF 34 MEDLINE
ACCESSION NUMBER: 91033507 MEDLINE
DOCUMENT NUMBER: 91033507 PubMed ID: 2227764
TITLE: [Rotation angioplasty of chronic **coronary artery stenosis**].
Rotationsangioplastik chronischer

Koronararterienverschlusse.
AUTHOR: Kaltenbach M; Vallbracht C
CORPORATE SOURCE: Abteilung fur Kardiologie, Johann-Wolfgang-Goethe-Universitat, Frankfurt am Main.
SOURCE: HERZ, (1990 Oct) 15 (5) 292-8.
Journal code: F88; 7801231. ISSN: 0340-9937.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199012
ENTRY DATE: Entered STN: 19910208
Last Updated on STN: 19910208
Entered Medline: 19901227

AB Coronary artery occlusion of more than six months duration can only rarely be recanalized with conventional techniques. For this reason, rotational angioplasty, which has been successfully applied for occlusion of peripheral arteries, has been employed in modified form for recanalization of chronic coronary artery occlusion. Rotational angioplasty is based on the concept that the slowly revolving, dull and relatively thick head of the flexible rotation catheter will seek the path of least resistance which, even in the case of relatively old arterial occlusions, mostly represents thrombotic material. The elastic, high-torque rotational catheter constructed of several V2A spiral steel wires has an interior lumen for insertion of exchange guidewires up to 0.014" and injection of contrast medium and an olive-shaped head of V2A steel with a diameter of 1.3 to 1.6 mm. A protection catheter made of polyethylene with metal markers and conically-tapered tip provides variable stiffness of the rotating catheter and protection of the endothelium in the proximal vascular segment. The slow rotation of 200 r.p.m. is performed with a small electric motor. Between April 1987 and February 1988, rotation angioplasty was performed in 20 patients, 17 with occlusion of the right coronary artery, two with occlusion of the left anterior descending artery and one with bypass graft occlusion to the left anterior descending artery in whom a conventional guidewire through the chronic occlusion could not be advanced. The duration of occlusion, based on previous angiograms anginal complaints or myocardial infarction, ranged from one month to twelve years, in twelve patients more than six months. In all patients, the indication for revascularization was clearly established.(ABSTRACT TRUNCATED AT 250 WORDS)

L9 ANSWER 28 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1989:489937 BIOSIS
DOCUMENT NUMBER: BA88:116474
TITLE: RESULTS OF PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY.
AUTHOR(S): ZEITLER E; FENG G; OLDENDORF M; RICHTER E-I; RITTER W; SEYFERTH W
CORPORATE SOURCE: CHEFARZT ABT. DIAGN. RADIOL. ZENTRUMS, STAEDTISCHES KLIN., FLURSTR. 17, D-8500 NUERNBERG.
SOURCE: HERZ, (1989) 14 (1), 22-28.
CODEN: HERZDW.
FILE SEGMENT: BA; OLD
LANGUAGE: German

AB Percutaneous transluminal angioplasty (PTA) can be subdivided into three epochs: 1. from its inception by Dotter and Judkins up to the first **coronary artery stenosis** dilatation with the Gruntzig balloon catheter system; 2. from the introduction of coronary stenosis dilatation by Gruntzig up to its unequivocal acceptance; 3. the period of influence of low-risk coronary dilatation on peripheral angioplasty and the search for techniques to compliment or obviate the need for balloon dilatation. The Gruntzig double-lumen balloon catheter system contributed to the lower rate of complications and higher success

rate. The clinical accepatance appeared greater for the coronary arteries since, in contrast to the peripheral vascular system, the indication for treatment is established by the physician performing the dilatation. PTA implies percutaneous puncture of a vessel with Seldinger technique and introduction of devices such as guidewires, Dotter and Gruntzig catheters among others, catheters with fiberglass for laser conduction and instruments for fractionating, drilling and cutting. The goal of PTA is to completely or partially eliminate, without surgery, intraluminal vascular narrowing in the presence of peripheral arterial disease in stage II, III or IV. Prerequisite to the use of PTA are: 1. adequate fluoroscopic and angiographic facilities; 2. adequate instrumentation; 3. experience with at least 200 procedures; 4. knowledge of the pathophysiology and adjunctive treatment; 5. knowledge of the treatment of complications; 6. cooperation with a vascular surgery service. A number of factors may influence the results of treatment. Adjunctive medical treatment: the use of platelet aggregation inhibitors and heparin influences the rate of early rethrombosis. Longterm anticoagulation has led to significantly more favorable patency rates up to three years after dilatation. A comparison of secondary phophylaxis with platelet aggregation inhibitors as compared with anticoagulation is not available. In iliac stenoses and short stenoses in the femoral artery region with good run-off, good longterm results can be achieved even without adjunctive medical treatment. Our patients are treated with **aspirin** 0.5 g or a combination of **aspirin** and dipyridamole three times daily. Run-off: longterm results after PTA in patients with previous stage III and IV depend to a large part on the number of freely patent lower leg arteries. In stage II, the influence can be detected, at best, as a trend. The prognosis after PTA in patients with femoro-popliteal obliteration in stages III and IV is comparable to that after surgical intervention. Age: for the primary and longterm results after PTA, the age is of lesser importance than the run-off. Comparison of the longterm results five years after successful PTA with respect to age showed statistically-significant differences only between those less than 50 and those older than 70 years. Stage of peripheral arterial disease, localization and length of occlusion: all of the latter factors can influence both the primary and longterm outcome. In a prospective study from 1976 to 1980, in 678 patients in stage III/IV and 1093 patients in stage IIb it was shown that not only in the presence of occlusion but also in stenotic lesions, angioplasty did not always lead to a primary success. A higher primary success rate has been rendered by guidewires with supersoft tips and steerability, pulsating guidewires as well as more accurate imaging techniques within the scope of digital subtraction angiography. The unsatisfactory results associated with femoro-popliteal obliteration with occlusion in excess of 10 cm and the only 30 to 40% recanalization rate of iliac occlusions provided the impetus for development of new modes of treatment. Recurrent stenoses; on deterioration of the walking distance or Doppler quotient, since recurrence can occur, angiography should be repeated. PTA, in any case, can be repeated. However, since attempts to recanalize the vessel after the second reccurence are met with a low success rate, alternative, new techniques may be taken into consideration as the primary intervention. New treatment techniques for PTA: Our experience encompasses the use of three laser application forms, the Kensey catheter system, pulsating guidewire systems, artherectomy and implantation of Strecker endovascular stents. Late results of up to one year are available. To date, however, indications and adjunctive medical treatment have not been uniformly agreed upon. Laser PTA: The goal of laser PTA has been to establish patency through long occlusions for subsequent balloon dilatation. Complete vaporization of the obliterative material is, however, conceivable. Dynamic angioplasty: the Kensey catheter system employs an instrument with a dull, rotating metal tip through which, with the aid of fluid infusion, the obliterating material is fractionated into small particles. In addition to heparin, the flushing fluid may also include

urokinase and a rheologically active substance. In clinical use, small emboli, in experimental studies in amputated limbs, large emboli have been observed. Nevertheless, the principle is promising. Other techniques with more slowly rotating instruments have been developed. Atherectomy: by means of the predominantly used Simpson excentric balloon catheter, an asymmetric atheroma formation can be pressed into a chamber and then severed with a rotating blade (rotation velocity 200 to 300 r.p.m.). The particles are collected in a peripheral chamber after which they can be studied histologically. Even in the presence of symmetrical stenoses and short occlusions, with no lesions to the wall, atherectomy has been performed. To date, favorable results have been observed. Endoprostheses: since the introduction of percutaneously implantable vascular prostheses by Dotter and Rabkin, new prostheses made of flexible mesh-wire have been produced which, if necessary, exhibit expansive properties. They have been tested for use in the iliac, femoral and coronary systems. The Walstent, Palmaz-Reuter stent and Strecker stent have been clinically tested. The indication for endoprosthesis is established primarily on the basis of recurrent stenosis or primary substantial residual stenosis. With these new techniques, without the risk of surgery, treatment is available for an increasing number of patients to maintain or improve quality of life and work capacity.

L9 ANSWER 29 OF 34 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 88322201 MEDLINE
DOCUMENT NUMBER: 88322201 PubMed ID: 3413717
TITLE: Epinephrine potentiation of in vivo stimuli reverses **aspirin** inhibition of platelet thrombus formation in stenosed canine coronary arteries.
AUTHOR: Folts J D; Rowe G G
CORPORATE SOURCE: Section of Cardiology, University of Wisconsin Hospital, Madison 53792.
CONTRACT NUMBER: HL 29586-04 (NHLBI)
SOURCE: THROMBOSIS RESEARCH, (1988 May 15) 50 (4) 507-16. Journal code: VRN; 0326377. ISSN: 0049-3848.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198810
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19881003

AB In 18 anesthetized dogs with a 70% mechanically produced **coronary artery stenosis**, blood flow measured with an electromagnetic flowmeter showed cyclical reductions in flow due to periodic acute platelet thrombus formation. These were abolished in eight of nine dogs with 2.5 mg/kg of **aspirin** given intravenously and in nine of nine dogs with 5 mg/kg of **aspirin**. However in 14 of 18 dogs the cyclical flow reductions were temporarily renewed with the infusion of epinephrine 0.4 microgram/kg/min. Human platelets inhibited with **aspirin** can be reactivated with physiologic amounts of epinephrine. We postulate that in patients with atherosclerotic stenotic lesions the use of **aspirin** to inhibit arterial thrombus formation may be less effective when they have elevated catecholamines.

L9 ANSWER 30 OF 34 MEDLINE
ACCESSION NUMBER: 88292418 MEDLINE
DOCUMENT NUMBER: 88292418 PubMed ID: 3041833
TITLE: Evolving concepts in the treatment of acute myocardial infarction.
AUTHOR: Lange R A; Hillis L D
CORPORATE SOURCE: Department of Internal Medicine, University of Texas

Southwestern Medical Center, Dallas 75235.
SOURCE: AMERICAN JOURNAL OF THE MEDICAL SCIENCES, (1988 Aug) 296 (2) 143-52. Ref: 62
Journal code: 3L2; 0370506. ISSN: 0002-9629.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198809
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 20000303
Entered Medline: 19880908

AB Recent studies in patients with transmural acute myocardial infarction have demonstrated that intravenous thrombolytic therapy with streptokinase or tissue plasminogen activator improves left ventricular function and reduces mortality. To accomplish this, these agents must be infused early, ie, within 3 to 4 hours of the onset of chest pain; later administration of the agents exerts no significant beneficial effect. Tissue plasminogen activator appears to be the most effective and safest of the available thrombolytic agents: its intravenous administration is followed by coronary reperfusion in about 70% of patients, and its use is not associated with allergic reactions, a systemic fibrinolytic state, or a prolonged fibrinolytic effect. Once reperfusion has been established with an intravenous thrombolytic agent, intravenous heparin is given for several days, followed by oral **aspirin** to prevent reocclusion. Since many of these patients have a residual high-grade **coronary artery stenosis** in the infarct-related artery, mechanical alleviation of the residual stenosis with angioplasty or bypass surgery is an attractive therapy 2 to 4 days after reperfusion, and preliminary data indicate that elective coronary angioplasty 3 days after thrombolytic therapy is beneficial. However, further studies are needed to assess more definitively the use of such an aggressive therapeutic strategy.

L9 ANSWER 31 OF 34 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 88048845 MEDLINE
DOCUMENT NUMBER: 88048845 PubMed ID: 2960295
TITLE: Laboratory test results as predictors of recurrent **coronary artery stenosis** following angioplasty.
AUTHOR: Austin G E; Lynn M; Hollman J
CORPORATE SOURCE: Atlanta Veterans Administration Medical Center, Decatur, GA 30333.
SOURCE: ARCHIVES OF PATHOLOGY AND LABORATORY MEDICINE, (1987 Dec) 111 (12) 1158-62.
Journal code: 79Z; 7607091. ISSN: 0003-9985.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198712
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19980206
Entered Medline: 19871217

AB A retrospective study has been conducted to examine the potential value of a battery of selected clinical laboratory tests as predictors of recurrent **coronary artery stenosis** following angioplasty. Data from 443 patients (including 325 men and 118 women) who had undergone coronary angioplasty were analyzed. Total men, total women, **aspirin**-treated men, and **aspirin**-treated women received

separate statistical treatment. The only statistically significant difference in mean laboratory values between success (no recurrent stenosis) and recurrence groups was for serum cholesterol in **aspirin**-treated women, where the recurrence group showed a higher value than the success group. Multiple logistic regression showed a statistically significant association between elevated mean cholesterol and low mean hemoglobin concentration and recurrence in the female **aspirin**-treated group. Although only a small number of the laboratory test results fell outside normal laboratory reference ranges, we noted that for some tests, patients with extreme values predominantly developed recurrent stenosis while for certain other tests they were mainly successful. For example, seven of eight male diabetics with plasma glucose concentrations above 9.4 mmol/L (170 mg/dL) developed recurrence, while recurrent stenosis did not occur in any of six men with a bleeding time greater than twice normal. The results of these studies do not support the hypothesis that lipoprotein, coagulation, and platelet factors influence the development of recurrent stenosis in the majority of patients, although abnormalities in certain of these parameters may contribute to the process in specific cases.

L9 ANSWER 32 OF 34 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1986:329790 BIOSIS
DOCUMENT NUMBER: BR31:44372
TITLE: CAN ENDOGENOUS PROSTACYCLIN CONTRIBUTE TO THE ANTITHROMBOTIC EFFECT OF LOW DOSE **ASPIRIN** IN-VIVO.
AUTHOR(S): WESELCOUCH E O; HUMPHREY W R; AIKEN J W
CORPORATE SOURCE: ATHEROSCLEROSIS AND THROMBOSIS RESEARCH, UPJOHN CO., KALAMAZOO, MICHIGAN 49001.
SOURCE: HAYAISHI, O. AND S. YAMAMOTO (ED.). ADVANCES IN PROSTAGLANDIN, THROMBOXANE, AND LEUKOTRIENE RESEARCH, VOL. 15. KYOTO CONFERENCE ON PROSTAGLANDINS, KYOTO, JAPAN, NOV. 1984. XXX+746P. RAVEN PRESS BOOKS, LTD.: NEW YORK, N.Y., USA. ILLUS, (1985) 0 (0), 513-516.
CODEN: ATLRD6. ISSN: 0732-8141. ISBN: 0-88167-113-4.
FILE SEGMENT: BR; OLD
LANGUAGE: English

L9 ANSWER 33 OF 34 MEDLINE
ACCESSION NUMBER: 84126337 MEDLINE
DOCUMENT NUMBER: 84126337 PubMed ID: 6320701
TITLE: Unstable rest angina with ST-segment depression. Pathophysiologic considerations and therapeutic implications.
AUTHOR: Oliva P B
SOURCE: ANNALS OF INTERNAL MEDICINE, (1984 Mar) 100 (3) 424-40.
Ref: 215
Journal code: 5A6; 0372351. ISSN: 0003-4819.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198403
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19840315
AB Because of recent findings, a reassessment is needed of the concept that rest angina associated with ST-segment depression is due to a spontaneous, transient increase of blood pressure or heart rate, or both, in the presence of critical **coronary artery stenosis**
. Continuous hemodynamic and electrocardiographic recordings done before

and during attacks of rest angina and thallium-201 scintigrams done during pain indicate that a transient reduction of flow is the immediate cause of ischemia in most, but not all, instances. Flow reduction, in turn, appears to be due to coronary arterial spasm or platelet aggregation, or both, acting at a site of atherosclerotic narrowing. Therapy for unstable rest angina should include measures to prevent both transient reductions of flow and increases of myocardial oxygen consumption. A combination of long-acting nitrates, a beta-blocker, a calcium-channel blocker, and **aspirin** or heparin is suggested for this purpose. Intravenous nitroglycerin is useful when angina occurs despite this therapy or when frequent attacks of ischemia are occurring at the time of admission.

L9 ANSWER 34 OF 34 MEDLINE
ACCESSION NUMBER: 83286793 MEDLINE
DOCUMENT NUMBER: 83286793 PubMed ID: 6224648
TITLE: Serial angiographic evidence of rapid resolution of **coronary artery stenosis.**
AUTHOR: Sanborn T A; Faxon D P; Kellett M A; Ryan T J
SOURCE: CHEST, (1983 Sep) 84 (3) 302-4.
Journal code: D1C; 0231335. ISSN: 0012-3692.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198310
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19831008

AB An example of rapid, spontaneous resolution of an eccentric coronary luminal narrowing from 95 percent to 80 percent and subsequently to 50 percent stenosis over a six-week time period is presented. Spontaneous thrombolysis is proposed as the explanation for these changes and is discussed with reference to existing experimental and clinical observations.

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(FILE 'HOME' ENTERED AT 17:15:24 ON 16 JAN 2002)

FILE 'MEDLINE, BIOSIS' ENTERED AT 17:15:37 ON 16 JAN 2002
L1 3785 S CORONARY(W)ARTERY(W)STENOSIS
L2 5 S L1 AND FISH(W)OIL?
L3 4 DUP REM L2 (1 DUPLICATE REMOVED)
L4 14 S L1 AND (DIET OR (LOWER? OR REDUC?)(W)(CHOLESTEROL OR FAT)(W)I
L5 10 DUP REM L4 (4 DUPLICATES REMOVED)
L6 2 S L1 AND NIACIN
L7 2 DUP REM L6 (0 DUPLICATES REMOVED)
L8 46 S L1 AND ASPIRIN
L9 34 DUP REM L8 (12 DUPLICATES REMOVED)

L11 ANSWER 1 OF 3 MEDLINE
ACCESSION NUMBER: 1999249773 MEDLINE
DOCUMENT NUMBER: 99249773 PubMed ID: 10235694
TITLE: Fluvastatin: a review of its use in lipid disorders.
COMMENT: Erratum in: Drugs 1999 Sep;58(3):404
AUTHOR: Langtry H D; Markham A
CORPORATE SOURCE: Adis International Limited, Mairangi Bay, Auckland, New Zealand.. demail@adis.co.nz
SOURCE: DRUGS, (1999 Apr) 57 (4) 583-606. Ref: 107
Journal code: EC2; 7600076. ISSN: 0012-6667.
PUB. COUNTRY: New Zealand
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990628
Last Updated on STN: 20000124
Entered Medline: 19990615

AB Fluvastatin is an HMG-CoA reductase inhibitor used to treat patients with hypercholesterolaemia. Since fluvastatin was last reviewed in Drugs, trials have shown its efficacy in the secondary prevention of coronary heart disease (CHD) events and death and have expanded knowledge of its effects in primary CHD prevention and its mechanisms of activity. In addition to reducing total (TC) and low density lipoprotein (LDL-C) cholesterol, fluvastatin has antiatherogenic, antithrombotic and antioxidant effects, can improve vascular function, and may have immunomodulatory effects. Although fluvastatin interacts with **bile acid sequestrants** (requiring separation of doses), its pharmacokinetics permit oral administration to most patient groups. Fluvastatin is well tolerated, with adverse effects usually mild and transient. Use of fluvastatin to reduce lipids in patients with primary hypercholesterolaemia is well established. Its effects are similar in most patient groups, with 20 to 80 mg/day reducing LDL-C by 22 to 36%, triglycerides (TG) by 12 to 18% and apolipoprotein B by 19 to 28% and increasing high density lipoprotein cholesterol by 3.3 to 5.6%. Attempts to find fluvastatin dosages with efficacy equivalent to that of other HMG-CoA reductase inhibitors produce variable results, but larger per-milligram fluvastatin dosages are needed when patients switch from other HMG-CoA reductase inhibitors. Combinations of fluvastatin with fibric acid derivatives and **bile acid sequestrants** produce additive effects. Small noncomparative studies suggest fluvastatin reduces LDL-C in patients with hypercholesterolaemia secondary to kidney disorders by < or = 40.5% and with type 2 diabetes mellitus by < or = 32%. Three large randomised, double-blind trials show fluvastatin can help prevent CHD events or death and slow disease progression in patients with CHD with or without hypercholesterolaemia. In the Fluvastatin Angiographic Restenosis trial in patients undergoing balloon angioplasty, fluvastatin 80 mg/day for 40 weeks reduced the postangioplasty rate of deaths plus myocardial infarctions (1.5% vs 4% with placebo, $p < 0.025$) without altering vessel luminal diameters. In the Lipoprotein and Coronary Atherosclerosis Study in patients with **coronary artery stenosis**, luminal diameter reduced to a significantly lesser extent after fluvastatin 20 mg twice daily than placebo after 2.5 years (-0.028 vs -0.01 mm, $p < 0.005$). The Lescol in Symptomatic Angina study found reductions in all cardiac events or cardiac death in patients after 1 year of fluvastatin 40 mg/day (1.6% vs 5.6% for placebo, $p < 0.05$). CONCLUSIONS: An evolving pattern of data suggests that, in addition to its well established efficacy and cost effectiveness in reducing

hypercholesterolaemia, fluvastatin may now also be considered for use in the secondary prevention of CHD.

L11 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1993:288990 BIOSIS
DOCUMENT NUMBER: PREV199345007115
TITLE: The effect on **coronary artery stenosis** of intensive pharmacologic step therapy to improve LDL and HDL in patients with normal plasma lipid levels.
AUTHOR(S): Sacks, Frank M. (1); Pasternak, Richard C.; Gibson, C. Michael; Rosner, Bernard; Stone, Peter H.
CORPORATE SOURCE: (1) Brigham and Women's Hosp., Boston, MA USA
SOURCE: Circulation, (1992) Vol. 86, No. 4 SUPPL. 1, pp. I743. Meeting Info.: 65th Scientific Sessions of the American Heart Association New Orleans, Louisiana, USA November 16-19, 1992
ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

L11 ANSWER 3 OF 3 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 91146414 MEDLINE
DOCUMENT NUMBER: 91146414 PubMed ID: 1997308
TITLE: [Xanthomas of the Achilles tendon as the cardinal symptom of sitolsterolemia].
Xanthome der Achillessehnen als Leitsymptom der Sitosterinamie.
AUTHOR: Grahlke B K
CORPORATE SOURCE: Marinesanitatszentrum, Flensburg.
SOURCE: DEUTSCHE MEDIZINISCHE WOCHENSCHRIFT, (1991 Mar 1) 116 (9) 335-8.
Journal code: ECL; 0006723. ISSN: 0012-0472.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199104
ENTRY DATE: Entered STN: 19910419
Last Updated on STN: 19910419
Entered Medline: 19910403
AB The circumference of both Achilles tendons had gradually increased over the years in a now 32-year-old man (diameter of the left tendon 4.5 cm, of the right one 3.5 cm). This finally led to exercise-related pain in both tendons. Biopsy revealed benign deposition of xanthomata. Serum total cholesterol concentration was 261 mg/dl. Determination of various sitosterol fragments in serum gave a beta-sitosterol level of 43 mg/dl (normal range 0.3-1.7 mg/dl), characteristic of sitosterolaemia, which is an autosomal recessive disease causing intestinal hyperabsorption of a range of plant steroids closely related to cholesterol. On a diet low in plant steroids and treatment with **cholestyramine** (up to 32 g daily) the beta-sitosterol concentration fell, but only to 35 mg/dl, because of poor patient compliance. The patient died suddenly from **coronary artery stenosis** seven months after the diagnosis of sitosterolaemia.

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(FILE 'HOME' ENTERED AT 17:15:24 ON 16 JAN 2002)

FILE 'MEDLINE, BIOSIS' ENTERED AT 17:15:37 ON 16 JAN 2002

L1	3785	S CORONARY(W)ARTERY(W)STENOSIS
L2	5	S L1 AND FISH(W)OIL?
L3	4	DUP REM L2 (1 DUPLICATE REMOVED)
L4	14	S L1 AND (DIET OR (LOWER? OR REDUC?)(W)(CHOLESTEROL OR FAT)(W)I
L5	10	DUP REM L4 (4 DUPLICATES REMOVED)
L6	2	S L1 AND NIACIN
L7	2	DUP REM L6 (0 DUPLICATES REMOVED)
L8	46	S L1 AND ASPIRIN
L9	34	DUP REM L8 (12 DUPLICATES REMOVED)
L10	4	S L1 AND (COLESTIPOL OR COLESTID OR QUESTRAN OR CHOLESTYRAMINE
L11	3	DUP REM L10 (1 DUPLICATE REMOVED)

L10 ANSWER 1 OF 6 USPATFULL
ACCESSION NUMBER: 2001:176240 USPATFULL
TITLE: Methods of treating alzheimer's disease
INVENTOR(S): Bisgaier, Charles L., Ann Arbor, MI, United States
Newton, Roger S., Ann Arbor, MI, United States

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2001028895	A1	20011011	
APPLICATION INFO.:	US 2001-776536	A1	20010202	(9)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2000-180406	20000204	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Patrea L Pabst Arnall Golden & Gregory LLP, 2800 One Atlantic Center, 1201 West Peachtree Street, Atlanta, GA, 30309-3450
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
LINE COUNT: 419
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Blood cholesterol levels are correlated with production of amyloid .beta. protein (A.beta.), and are predictors of populations at risk of developing AD. Methods for increasing HDL-cholesterol levels, HDL-apoA-I levels, or HDL function, can be used to decrease production of A.beta., thereby decreasing the risk of developing AD. Compounds which function as HDL include synthetic HDL which contains lipid such as phosphotidyl choline, phosphatidyl serine, phosphatidyl ethanolamine, and other phospholipids. Compounds which enhance HDL function include HDL associated proteins such as apo A1 or variants thereof including apo AI-Milano and biologically active peptides derived therefrom, reverse lipid transport (RLT) peptides, apoE, enzymes associated with HDL such as paraoxonase, and LCAT, alone or, more preferably, formulated in combination with liposomes or emulsions. These compositions can also be administered with compounds that increase HDL levels specifically, and thereby improve the HDL cholesterol to total cholesterol ratio or the apoA-I to total cholesterol ratio, and/or with compositions which are effective to improve the HDL or apoA-I to total blood cholesterol levels. Alternatively, or in addition, cholesteryl ester transfer protein inhibitors (CETP inhibitors) can be administered to the patients to treat or prevent Alzheimer's.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 2 OF 6 USPATFULL
ACCESSION NUMBER: 2001:136225 USPATFULL
TITLE: Method of inhibiting 5.alpha.-reductase with astaxanthin
INVENTOR(S): Anderson, Mark, Carmel, NY, United States
PATENT ASSIGNEE(S): Triarco Industries, Inc., Wayne, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6277417	B1	20010821	
APPLICATION INFO.:	US 2000-546316		20000407	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Tate, Christopher R.

LEGAL REPRESENTATIVE: Fitzpatrick, Cella, Harper & Scinto
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
LINE COUNT: 590
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for inhibiting the activity of the enzyme 5.alpha.-reductase in a human subject is provided which comprises administering to the subject a composition comprising the carotenoid astaxanthin. Administration of the composition to inhibit the enzyme is useful to prevent and treat benign prostate hyperplasia (BPH) and prostate cancer in human males.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 3 OF 6 CAPLUS
ACCESSION NUMBER: 2001:581714 CAPLUS
DOCUMENT NUMBER: 135:147450
TITLE: Methods for treating Alzheimer's disease by lowering blood cholesterol levels
INVENTOR(S): Bisgaier, Charles; Newton, Roger S.
PATENT ASSIGNEE(S): Esperion Therapeutics Inc., USA
SOURCE: PCT Int. Appl., 18 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001056579	A1	20010809	WO 2001-US3580	20010202
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2001028895	A1	20011011	US 2001-776536	20010202

PRIORITY APPLN. INFO.: US 2000-180406 P 20000204
AB Blood cholesterol levels are correlated with prodn. of amyloid .beta. protein (A.beta.), and risk of developing AD. Increasing HDL-cholesterol levels, HDL-apoA-I levels, or HDL function, decrease prodn. of A.beta.. Compds. which function as HDL include synthetic HDL which contains lipid such as phosphatidyl choline, phosphatidyl serine, phosphatidyl ethanolamine. Compds. which enhance HDL function include HDL assocd. proteins such as apo A1 or variants, reverse lipid transport peptides, apoE, enzymes assocd. with HDL such as paraoxonase, and LCAT, preferably, formulated in combination with liposomes or emulsions. These compns. can also be administered with compds. that increase HDL levels specifically, and thereby improve the HDL cholesterol to total cholesterol ratio or the apoA-I to total cholesterol ratio, and/or with compns. which are effective to improve the HDL or apoA-I to total blood cholesterol levels. Alternatively, cholesteryl ester transfer protein inhibitors can be used to treat Alzheimer's.
REFERENCE COUNT: 11
REFERENCE(S): (1) Childrens Medical Center; WO 9948488 A 1999 CAPLUS
(7) Medical Res Council; WO 9506456 A 1995 CAPLUS
(8) Strittmatter, W; WO 9908701 A 1999 CAPLUS
(9) Univ British Columbia; WO 9523592 A 1995 CAPLUS
(10) Warner Lambert Co; WO 9938498 A 1999 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 4 OF 6 USPATFULL
ACCESSION NUMBER: 2000:168040 USPATFULL
TITLE: Methods and compositions for the rapid and enduring relief of inadequate myocardial function
INVENTOR(S): Seed, Brian, Boston, MA, United States
Seed, John C., Princeton, NJ, United States
PATENT ASSIGNEE(S): Heart Care Partners, Princeton, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6159993		20001212
APPLICATION INFO.:	US 1998-198874		19981124 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1996-680684, filed on 17 Jul 1996, now patented, Pat. No. US 5861399
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jordan, Kimberly
LEGAL REPRESENTATIVE: Clark & Elbing LLP
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
LINE COUNT: 993
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are methods and compositions for reducing coronary artery stenosis, restoring blood flow to infarcted myocardium, improving myocardial perfusion, reducing heart attacks or other adverse cardiovascular events, or treating symptoms of inadequate myocardial function in a mammal involving administering to the mammal (a) a compound that includes eicosapentaeneoic acid or docosahexaeneoic acid and (b) a cholesterol-lowering therapeutic, combined with dietary restrictions (resulting in aggressive loading of marine lipids), whereby a serum LDL concentration of less than 75 mg/dl (and preferably less than 55 mg/dl) is achieved. One particular method involves administering to the mammal a combination that includes (a) a compound that includes an eicosapentaeneoic or docosahexaeneoic acid (for example, a marine lipid) and (b) a cholesterol synthesis or transfer inhibitor, and which may also optionally include aspirin and/or niacin. The methods and compositions of the invention may also further include a bile acid sequestrant and/or buspirone. Also disclosed are methods for treating heart disease that involve administration of buspirone.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 5 OF 6 USPATFULL
ACCESSION NUMBER: 1999:7386 USPATFULL
TITLE: Methods and compositions for the rapid and enduring relief of inadequate myocardial function
INVENTOR(S): Seed, Brian, Boston, MA, United States
Seed, John C., Princeton, NJ, United States
PATENT ASSIGNEE(S): Heart Care Partners, Princeton, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5861399		19990119
APPLICATION INFO.:	US 1996-680684		19960717 (8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jordan, Kimberly
LEGAL REPRESENTATIVE: Clark & Elbing LLP

NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1
LINE COUNT: 854
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed are methods and compositions for reducing coronary artery stenosis, restoring blood flow to infarcted myocardium, improving myocardial perfusion, reducing heart attacks or other adverse cardiovascular events, or treating symptoms of inadequate myocardial function in a mammal involving administering to the mammal (a) a compound that includes eicosapentaeneoic acid or docosahexaeneoic acid and (b) a cholesterol-lowering therapeutic, combined with dietary restrictions (resulting in aggressive loading of marine lipids), whereby a serum LDL concentration of less than 75 mg/dl (and preferably less than 55 mg/dl) is achieved. One particular method involves administering to the mammal a combination that includes (a) a compound that includes an eicosapentaeneoic or docosahexaeneoic acid (for example, a marine lipid) and (b) a cholesterol synthesis or transfer inhibitor, and which may also optionally include aspirin and/or niacin. The methods and compositions of the invention may also further include a bile acid sequestrant and/or buspirone. Also disclosed are methods for treating heart disease that involve administration of buspirone.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:495161 CAPLUS
DOCUMENT NUMBER: 131:125474
TITLE: Method for treating Alzheimer's disease with agents lowering plasma triglycerides and optional hypocholesterolemic agents
INVENTOR(S): Bisgaier, Charles Larry; Emmerling, Mark Richard
PATENT ASSIGNEE(S): Warner-Lambert Company, USA
SOURCE: PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9938498	A1	19990805	WO 1998-US25495	19981202
W: AL, AU, BA, BB, BG, BR, CA, CN, CU, CZ, EE, GE, HR, HU, ID, IL, IS, JP, KP, KR, LC, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL, RO, SG, SI, SK, SL, TR, TT, UA, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9916165	A1	19990816	AU 1999-16165	19981202
BR 9814923	A	20001017	BR 1998-14923	19981202
EP 1051161	A1	20001115	EP 1998-960605	19981202
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1998-72912 P 19980128
WO 1998-US25495 W 19981202
OTHER SOURCE(S): MARPAT 131:125474

AB A method for treating or preventing the onset of Alzheimer's Disease comprises administering to a mammal in need thereof an Alzheimer's Disease-preventing or -treating amt. of a plasma triglyceride level-lowering agent. Optionally, the plasma triglyceride level-lowering agent can be co-administered with a cholesterol level-lowering agent. The

relationship between Alzheimer's disease and known risk factors for cardiovascular disease was also studied.

REFERENCE COUNT: 15

REFERENCE(S):
(1) Esmond, R; WO 9839967 A 1998 CAPLUS
(3) Horrobin, D; WO 9816216 A 1998 CAPLUS
(4) Innovative Tech Center; WO 9220335 A 1992 CAPLUS
(5) Kanagawa Kagaku Kenkyujyo Kk; JP 08143454 A 1996 CAPLUS
(6) Leininger-Muller, B; LIFE SCIENCES 1996, V58(6), P455 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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(FILE 'HOME' ENTERED AT 14:28:17 ON 16 JAN 2002)

FILE 'REGISTRY' ENTERED AT 14:28:23 ON 16 JAN 2002
E EICOSAPENTA?/CN
L1 3 S E10
E DOCOSAHEXA?/CN
L2 3 S E12
E ASPIRIN/CN
L3 1 S E3
E NIACIN/CN
L4 1 S E3

FILE 'MEDLINE, BIOSIS, USPATFULL, CAPLUS' ENTERED AT 14:33:09 ON 16 JAN 2002
L5 49 S (L1 OR L2) AND L4
L6 45 DUP REM L5 (4 DUPLICATES REMOVED)
L7 77 S (L1 OR L2) AND L3
L8 70 DUP REM L7 (7 DUPLICATES REMOVED)
L9 6 S L6 AND (?STATIN? OR REDUCTASE(W)INHIBITO? OR QUESTRAN OR COL
L10 6 DUP REM L9 (0 DUPLICATES REMOVED)
L11 5 S L8 AND (?STATIN? OR REDUCTASE(W)INHIBITO? OR QUESTRAN OR COL

L11 ANSWER 1 OF 5 USPATFULL
ACCESSION NUMBER: 2001:36869 USPATFULL
TITLE: Methods for treating neurotransmitter-mediated pain syndromes by topically administering an omega fatty acid
INVENTOR(S): Mease, Philip J., Seattle, WA, United States
Bockow, Barry I., Seattle, WA, United States
Erlitz, Marc D., Kirkland, WA, United States
PATENT ASSIGNEE(S): MyoRx, Inc., Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6201022	B1	20010313
APPLICATION INFO.:	US 1997-824931		19970327 (8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jarvis, William R. A.
LEGAL REPRESENTATIVE: Seed Intellectual Property Law Group PLLC
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
LINE COUNT: 655
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are disclosed for treating neurotransmitter-mediated pain syndromes such as fibromyalgia. Such methods include topically administering an effective amount of a composition containing an omega fatty acid in combination with a carrier or diluent. The composition may also contain a cyclo-oxygenase inhibitor and other optional components, such as vitamins A, E and/or C.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 2 OF 5 USPATFULL
ACCESSION NUMBER: 2000:168040 USPATFULL
TITLE: Methods and compositions for the rapid and enduring relief of inadequate myocardial function
INVENTOR(S): Seed, Brian, Boston, MA, United States
Seed, John C., Princeton, NJ, United States
PATENT ASSIGNEE(S): Heart Care Partners, Princeton, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6159993		20001212
APPLICATION INFO.:	US 1998-198874		19981124 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1996-680684, filed on 17 Jul 1996, now patented, Pat. No. US 5861399
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jordan, Kimberly
LEGAL REPRESENTATIVE: Clark & Elbing LLP
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
LINE COUNT: 993
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are methods and compositions for reducing coronary artery stenosis, restoring blood flow to infarcted myocardium, improving myocardial perfusion, reducing heart attacks or other adverse cardiovascular events, or treating symptoms of inadequate myocardial function in a mammal involving administering to the mammal (a) a compound that includes eicosapentaeneoic acid or docosahexaeneoic acid

and (b) a cholesterol-lowering therapeutic, combined with dietary restrictions (resulting in aggressive loading of marine lipids), whereby a serum LDL concentration of less than 75 mg/dl (and preferably less than 55 mg/dl) is achieved. One particular method involves administering to the mammal a combination that includes (a) a compound that includes an eicosapentaeneoic or docosahexaeneoic acid (for example, a marine lipid) and (b) a cholesterol synthesis or transfer inhibitor, and which may also optionally include aspirin and/or niacin. The methods and compositions of the invention may also further include a bile acid sequestrant and/or buspirone. Also disclosed are methods for treating heart disease that involve administration of buspirone.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 3 OF 5 USPATFULL

ACCESSION NUMBER:	2000:121554 USPATFULL
TITLE:	Compounds and therapies for the prevention of vascular and non-vascular pathologies
INVENTOR(S):	Grainger, David J., Cambridge, United Kingdom Metcalfe, James C., Cambridge, United Kingdom Kasina, Sudhakar, Mercer Island, WA, United States
PATENT ASSIGNEE(S):	NeoRx Corporation, Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6117911		20000912	
APPLICATION INFO.:	US 1998-57323		19980409	(9)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 1997-43852	19970411	(60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Lambkin, Deborah C.
LEGAL REPRESENTATIVE:	Schwegman, Lundberg, Woessner & Kluth, P.A.
NUMBER OF CLAIMS:	18
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	13 Drawing Figure(s); 14 Drawing Page(s)
LINE COUNT:	4129

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides a method of treating a mammal having, or at risk of, an indication associated with a TGF-beta deficiency comprising administering one or more agents that is effective to elevate the level of TGF-beta. The invention also provides novel compounds that elevate TGF-beta levels, as well as pharmaceutical compositions comprising compounds that elevate TGF-beta levels, and methods for detecting diseases associated with endothelial cell activation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 4 OF 5 USPATFULL

ACCESSION NUMBER:	1999:7386 USPATFULL
TITLE:	Methods and compositions for the rapid and enduring relief of inadequate myocardial function
INVENTOR(S):	Seed, Brian, Boston, MA, United States Seed, John C., Princeton, NJ, United States
PATENT ASSIGNEE(S):	Heart Care Partners, Princeton, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE

PATENT INFORMATION: US 5861399 19990119
APPLICATION INFO.: US 1996-680684 19960717 (8)
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jordan, Kimberly
LEGAL REPRESENTATIVE: Clark & Elbing LLP
NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1
LINE COUNT: 854
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed are methods and compositions for reducing coronary artery stenosis, restoring blood flow to infarcted myocardium, improving myocardial perfusion, reducing heart attacks or other adverse cardiovascular events, or treating symptoms of inadequate myocardial function in a mammal involving administering to the mammal (a) a compound that includes eicosapentaeneoic acid or docosahexaeneoic acid and (b) a cholesterol-lowering therapeutic, combined with dietary restrictions (resulting in aggressive loading of marine lipids), whereby a serum LDL concentration of less than 75 mg/dl (and preferably less than 55 mg/dl) is achieved. One particular method involves administering to the mammal a combination that includes (a) a compound that includes an eicosapentaeneoic or docosahexaeneoic acid (for example, a marine lipid) and (b) a cholesterol synthesis or transfer inhibitor, and which may also optionally include aspirin and/or niacin. The methods and compositions of the invention may also further include a bile acid sequestrant and/or buspirone. Also disclosed are methods for treating heart disease that involve administration of buspirone.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:708808 CAPLUS
DOCUMENT NUMBER: 129:310911
TITLE: TGF-.beta.-elevating compounds and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods
INVENTOR(S): Grainger, David J.; Metcalfe, James C.; Kasina, Sudhakar
PATENT ASSIGNEE(S): Neorx Corp., USA
SOURCE: PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9846588	A2	19981022	WO 1998-US7063	19980409
WO 9846588	A3	19990107		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9869598	A1	19981111	AU 1998-69598	19980409
US 6117911	A	20000912	US 1998-57323	19980409

PRIORITY APPLN. INFO.: US 1997-43852 P 19970411
WO 1998-US7063 W 19980409

OTHER SOURCE(S): MARPAT 129:310911
AB A method is provided for treating a mammal having, or at risk of, an indication assocd. with a TGF-.beta. deficiency, comprising administering one or more agents that is effective to elevate the level of TGF-.beta.. The invention also provides compds. that elevate TGF-beta levels, as well as pharmaceutical compns. comprising compds. that elevate TGF-beta levels and methods for detecting diseases assocd. with endothelial cell activation.

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(FILE 'HOME' ENTERED AT 14:28:17 ON 16 JAN 2002)

FILE 'REGISTRY' ENTERED AT 14:28:23 ON 16 JAN 2002
E EICOSAPENTA?/CN
L1 3 S E10
E DOCOSAHEXA?/CN
L2 3 S E12
E ASPIRIN/CN
L3 1 S E3
E NIACIN/CN
L4 1 S E3

FILE 'MEDLINE, BIOSIS, USPATFULL, CAPLUS' ENTERED AT 14:33:09 ON 16 JAN 2002
L5 49 S (L1 OR L2) AND L4
L6 45 DUP REM L5 (4 DUPLICATES REMOVED)
L7 77 S (L1 OR L2) AND L3
L8 70 DUP REM L7 (7 DUPLICATES REMOVED)
L9 6 S L6 AND (?STATIN? OR REDUCTASE(W)INHIBITO? OR QUESTRAN OR COL
L10 6 DUP REM L9 (0 DUPLICATES REMOVED)
L11 5 S L8 AND (?STATIN? OR REDUCTASE(W)INHIBITO? OR QUESTRAN OR COL
L12 2 S L10 AND ?STENOSI?
L13 4 S L11 AND ?STENOSI?

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L12  2 ANSWERS  USPATFULL
AN     2000:168040  USPATFULL
TI     Methods and compositions for the rapid and enduring relief of inadequate
       myocardial function
NCL    NCLM:  514/356.000
IC     [7]
       ICM: A61K031-20
       ICS: A61K031-04
GI     SECTION      PAGES      FORMAT     SIZE
       -----------  ---------  ---------  -------
       FRONT PAGE   1          PAGE.FP    39K
       DESCRIPTION  2-8        PAGE.DESC  991K
       CLAIMS       8-9        PAGE.CLM   210K
       COMPLETE     1-9        PAGE.ALL   1111K

       Use PAGE(n) to retrieve a specific page

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L12  2 ANSWERS  USPATFULL
AN     1999:7386  USPATFULL
TI     Methods and compositions for the rapid and enduring relief of inadequate
       myocardial function
NCL    NCLM:  514/252.150
       NCLS:  514/356.000; 514/419.000; 514/451.000; 514/460.000; 514/510.000;
              514/548.000; 514/560.000; 514/741.000; 514/824.000
IC     [6]
       ICM: A61K031-495
       ICS: A61K031-50; A61K031-44; A61K031-40
GI     SECTION      PAGES      FORMAT     SIZE
       -----------  ---------  ---------  -------
       FRONT PAGE   1          PAGE.FP    39K
       DESCRIPTION  2-8        PAGE.DESC  942K
       CLAIMS       8-8        PAGE.CLM   122K
       COMPLETE     1-8        PAGE.ALL   982K

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ALL ANSWERS HAVE BEEN SCANNED


=> d scan l13

L13  4 ANSWERS  USPATFULL
AN     2000:121554  USPATFULL
TI     Compounds and therapies for the prevention of vascular and non-vascular
       pathologies
NCL    NCLM:  514/648.000
       NCLS:  564/317.000
IC     [7]
       ICM: A61K031-135
       ICS: C07C213-00
GI     SECTION      PAGES      FORMAT     SIZE
       -----------  ---------  ---------  -------
       FRONT PAGE   1-3        PAGE.FP    223K
       DRAWINGS     4-17       PAGE.DRAW  187K
       DESCRIPTION  18-49      PAGE.DESC  3728K
       CLAIMS       49-54      PAGE.CLM   503K
       COMPLETE     1-54       PAGE.ALL   4519K

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HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):4

L13 4 ANSWERS USPATFULL
AN 2000:168040 USPATFULL
TI Methods and compositions for the rapid and enduring relief of inadequate myocardial function
NCL NCLM: 514/356.000
IC [7]
ICM: A61K031-20
ICS: A61K031-04
GI

SECTION	PAGES	FORMAT	SIZE
FRONT PAGE	1	PAGE.FP	39K
DESCRIPTION	2-8	PAGE.DESC	991K
CLAIMS	8-9	PAGE.CLM	210K
COMPLETE	1-9	PAGE.ALL	1111K

Use PAGE(n) to retrieve a specific page

L13 4 ANSWERS CAPLUS COPYRIGHT 2002 ACS
IC ICM C07D333-38
ICS A61K031-60; A61K031-135; G01N033-543
CC 1-12 (Pharmacology)
Section cross-reference(s): 15, 63
TI TGF-.beta.-elevating compounds and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods
ST TGFbeta stimulating compd therapeutic; endothelial cell activation disease diagnosis
IT Transcription factors
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(I.kappa.B (inhibitor of NF-.kappa.B), .alpha.; TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)
IT Intracellular transport
(NF-.kappa.B translocation to nucleus; TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)
IT Cell nucleus
(NF-.kappa.B translocation to; TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)
IT High-density lipoproteins
Lipoproteins
Low-density lipoproteins
Very low-density lipoproteins
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(TGF-.beta. assocn. with lipoprotein particles)
IT Animal cells
(TGF-.beta. type II receptor-contg. mammalian cell detection; TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)
IT Anti-Alzheimer's drugs
Antiatherosclerotics
Anticholesteremic agents
Antiosteoporotic agents
Antiparkinsonian agents
Antiproliferative agents
Antirheumatic drugs
Autoimmune diseases
Blood analysis
Body fluid

Cardioprotectants
Cell proliferation
Chylomicrons
Coronary artery **stenosis**
Diabetes mellitus
Drug delivery systems
Fibrosis
Hypertriglyceridemia
Hypolipemic agents
Immunoassay
Lupus erythematosus
Marfan syndrome
Multiple sclerosis
Red wine
Senile dementia
Synergistic drug interactions
Vascular diseases
Vascular smooth muscle
(TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT Transforming growth factor .alpha.
Tumor necrosis factor .alpha.
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT Fish oils
Omega-3 fatty acids
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT Antibodies
IgD
IgG
IgG2
Immunoglobulins
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT Glycerides, biological studies
Osteopontin
.alpha.-Actins
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT Estrogen receptors
NF-.kappa.B
Protein HSP90
Transforming growth factor .beta. receptors
Transforming growth factor .beta. type II receptors
Transforming growth factors .beta.
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT Diagnosis
(endothelial cell activation-assocd. disease; TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT Vascular endothelium

(endothelial cell activation; TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT Atherosclerosis
(plaque stability; TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT Drug delivery systems
(unit doses; TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT **50-78-2**, Aspirin **50-78-2D**, Aspirin, derivs. 67-98-1, MER25 493-53-8 7440-50-8D, Copper, aspirinates 10540-29-1, Tamoxifen 23325-63-5 **32839-18-2**, Docosahexaenoic acid **32839-30-8**, Eicosapentaenoic acid 79902-63-9, **Simvastatin** 146063-51-6
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

IT 57-88-5, Cholesterol, biological studies
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(TGF-.beta.-elevating compds. and therapies for the prevention of vascular and non-vascular pathologies, and diagnostic methods)

L13 4 ANSWERS USPATFULL
AN 1999:7386 USPATFULL
TI Methods and compositions for the rapid and enduring relief of inadequate myocardial function
NCL NCLM: 514/252.150
NCLS: 514/356.000; 514/419.000; 514/451.000; 514/460.000; 514/510.000; 514/548.000; 514/560.000; 514/741.000; 514/824.000
IC [6]
ICM: A61K031-495
ICS: A61K031-50; A61K031-44; A61K031-40
GI

SECTION	PAGES	FORMAT	SIZE
FRONT PAGE	1	PAGE.FP	39K
DESCRIPTION	2-8	PAGE.DESC	942K
CLAIMS	8-8	PAGE.CLM	122K
COMPLETE	1-8	PAGE.ALL	982K

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☐ 1: Control Clin Trials 1987 Dec;8(4):356-87 Related Articles, NEW Books, LinkOut

The Cholesterol Lowering Atherosclerosis Study (CLAS): design, methods, and baseline results.

Blankenhorn DH, Johnson RL, Nessim SA, Azen SP, Sanmarco ME, Selzer RH.

The Cholesterol Lowering Atherosclerosis Study (CLAS) is a prospective, placebo-controlled, angiographic trial designed to test the hypothesis that aggressive lowering of LDL cholesterol with concomitant increase in HDL cholesterol will reverse or retard the atherosclerotic process. Specifically, CLAS was designed to determine whether combined therapy with colestipol plus niacin will produce clinically significant change in coronary, carotid, and femoral artery atherosclerosis and coronary bypass graft lesions. To this purpose, 188 subjects were randomized to diet plus drug or diet plus placebo. We report on methodological aspects of planning and evaluating this study, including the choice of the study population, procedures for recruitment, the experimental design including sample size considerations, methods for evaluating outcome, and methods for evaluating compliance to treatment. Comparison of baseline data indicated no significant differences between groups at the time of randomization. Subjects were predominantly male, Caucasian, 54 years of age, 20% above ideal weight, with normal blood pressure. The average age at bypass was 50 years. The average lipids were cholesterol (243 mg/dL), HDL (45 mg/dL), and LDL (168 mg/dL). Finally, the distribution of baseline coronary stenosis was equivalent between the two groups (average number of lesions per subject = 10.6).

Publication Types:
- Clinical Trial
- Randomized Controlled Trial

PMID: 3327654 [PubMed - indexed for MEDLINE]

Fish oil CAS.

L3 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:268982 BIOSIS
DOCUMENT NUMBER: BA92:1597
TITLE: EPA IN THE PREVENTION OF RESTENOSIS POST PTCA.
AUTHOR(S): BOWLES M H; KLONIS D; PLAVAC T G; GONZALES B; FRANCISCO D A; ROBERTS R W; BOXBERGER G R; POLINER L R; GALICHIA J P
CORPORATE SOURCE: 551 N. HILLSIDE, STE. 410, WICHITA, KANSAS 67214.
SOURCE: ANGIOLOGY, (1991) 42 (3), 187-194.
CODEN: ANGIAB. ISSN: 0003-3197.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB The effect of **fish oil** on restenosis was evaluated in patients undergoing coronary balloon angioplasty. In addition to routine pharmacotherapy, subjects were given 2.8 g of eicosapentanoic acid (EPA) daily. Treatment was started within twenty-four hours after successful percutaneous transluminal coronary angioplasty (PTCA). After six months of therapy, participants were subjected to coronary arteriography, exercise scintigraphy, exercise electrocardiography, or clinical evaluation. Follow-up evaluation involved 97 coronary lesions in 85 patients. Partial or singificant restenosis occurred in 36.5% of patients and 33% of vessels. The presence of severe stenosis before PTCA, dissection, thrombus, multilesion PTCA, and template bleeding time values were not correlated with restenosis. Dilation of the left anterior descending (LAD) and a residual stenosis .gtoreq. 35% were associated with restenosis. Approximately 20% of the patients related difficulty in taking the **fish oil**. Furthermore, these results show no advantage over expected restenosis rates.

L3 ANSWER 2 OF 4 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 89149869 MEDLINE
DOCUMENT NUMBER: 89149869 PubMed ID: 2920067
TITLE: Does platelet aggregation play a role in the reduction in localized intimal proliferation in normolipidemic pigs with fixed **coronary artery stenosis** fed dietary **fish oil**?.
AUTHOR: Hartog J M; Lamers J M; Essed C E; Schalkwijk W P; Verdouw P D
CORPORATE SOURCE: Laboratory for Experimental Cardiology, Erasmus University Rotterdam, The Netherlands.
SOURCE: ATHEROSCLEROSIS, (1989 Mar) 76 (1) 79-88.
Journal code: 95X; 0242543. ISSN: 0021-9150.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198904
ENTRY DATE: Entered STN: 19900306
Last Updated on STN: 19980206
Entered Medline: 19890404
AB In order to investigate the effect of **fish oil** on intimal proliferation of coronary arteries with a fixed stenosis normolipidemic piglets received a basic diet to which either 9% (w/w) lard (L, n = 8) or 4.5% (w/w) lard and 4.5% (w/w) mackerel oil (ML, n = 8) was added for 4 months. Stenosis was applied by implanting a 4.0 X 2.0 mm (i.d.) Teflon constrictor around the left anterior descending coronary artery (LADCA) (o.d. 2.7 +/- 0.1 mm). During the dietary period ADP-induced platelet aggregation in whole blood was higher in L than in ML. Partial replacement of 20:4 n - 6 by 20:5 n - 3 fatty acids in the platelet membranes of ML may have altered platelet aggregation by changes in eicosanoid synthesis. The plasma cholesterol and triglyceride levels did not change in L, but decreased in ML. At the end of the 4-month

dietary period the animals were again anesthetized and regional myocardial perfusion (radioactive labelled microspheres) and systolic segment length shortening (SLS) were measured while the hearts were paced at 160 pulses/min. Perfusion and SLS of non-LADCA nourished segment were similar for L and ML. However, transmural flow to the LADCA perfused myocardium was impaired in both groups, but the deficiency in endocardial perfusion was considerably larger in L than in ML, resulting in a larger loss of SLS in the former. Remote (2-3 cm from the site of the constrictor) luminal encroachment was minimal (less than 2%) in both groups, but at the site of the constrictor there was significant encroachment in both groups which was higher in L (62 +/- 7%) than in ML (11 +/- 4%). It is thought that in these normolipidemic pigs the reduction in platelet aggregation may play a role in the smaller intimal proliferation of the **fish oil**-fed animals.

L3 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1989:239053 BIOSIS
DOCUMENT NUMBER: BR36:117537
TITLE: DIETARY **FISH OIL** REDUCES INTIMAL PROLIFERATION OF THE CORONARY ARTERY CAUSED BY IMPLANTATION OF A CONSTRICTOR IN PIGS.
AUTHOR(S): LAMERS J M J; HARTOG J M; VERDOUW P D
CORPORATE SOURCE: DEP. BIOCHEM. I, THORAXCENTRE, ERASMUS UNIV. ROTTERDAM, ROTTERDAM, NETHERLANDS.
SOURCE: FIFTH INTERNATIONAL SYMPOSIUM ON EICOSANOIDS IN THE CARDIOVASCULAR SYSTEM, HALLE (SAALE), EAST GERMANY, MAY 16-19, 1988. BIOMED BIOCHIM ACTA, (1988) 47 (10-11), S83-S85.
CODEN: BBIADT. ISSN: 0232-766X.
FILE SEGMENT: BR; OLD
LANGUAGE: English

L3 ANSWER 4 OF 4 MEDLINE
ACCESSION NUMBER: 81099607 MEDLINE
DOCUMENT NUMBER: 81099607 PubMed ID: 7454129
TITLE: Pathophysiology of long-chain polyene fatty acids in heart muscle.
AUTHOR: Gudbjarnason S
SOURCE: NUTRITION AND METABOLISM, (1980) 24 Suppl 1 142-6.
Journal code: OAT; 0330472. ISSN: 0029-6678.
PUB. COUNTRY: Switzerland
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198103
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810324

AB The polyene fatty acid composition of cardiac phospholipids is modified by age, diet and stress in the rat. In the aging heart there is a progressive replacement of 18:2n6 by 20:4n6 in phosphatidylcholine (PC) and a replacement of 18:2n6 by 22:6n3 in phosphatidylethanolamine (PE). Norepinephrine stress accelerates aging of cardiac PC and PE. Dietary **fish oil** causes a replacement of 18:2n6 and 20:4n6 by 22:6n3 in cardiac PC and PE but not in cardiolipin. Studies on human cardiac autopsy samples suggest that: (a) polyene fatty acid composition changes with age; (b) stability of cardiac phospholipids is a function of the fatty acid composition, chain length and unsaturation; (c) coronary atherosclerosis is associated with a reduced content of 18:2n6 in phospholipids, an increased content of glycerides of abnormal composition and an unexpectedly low level of free fatty acids (FFA) in the heart muscle, and (d) many cases of sudden cardiac death in the absence of

marked **coronary artery stenosis** or
myocardial infarction may be associated with significant alterations in
myocardial levels of FFA (increase) or PE (decrease).

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(FILE 'HOME' ENTERED AT 17:15:24 ON 16 JAN 2002)

FILE 'MEDLINE, BIOSIS' ENTERED AT 17:15:37 ON 16 JAN 2002

L1	3785	S CORONARY(W)ARTERY(W)STENOSIS
L2	5	S L1 AND FISH(W)OIL?
L3	4	DUP REM L2 (1 DUPLICATE REMOVED)

L5 ANSWER 1 OF 10 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1995:505855 BIOSIS
DOCUMENT NUMBER: PREV199598510905
TITLE: The influence of pretreatment low density lipoprotein cholesterol concentrations on the effect of hypocholesterolemic therapy on coronary atherosclerosis in angiographic trials.
AUTHOR(S): Sacks, Frank M. (1); Gibson, C. Michael; Rosner, Bernard; Pasternak, Richard C.; Stone, Peter H.; Group, The Harvard Atherosclerosis Reversibility Project Research
CORPORATE SOURCE: (1) Nutrition Dep., Harv. Sch. Public Health, 655 Huntington Ave., Boston, MA 02115 USA
SOURCE: American Journal of Cardiology, (1995) Vol. 76, No. 9, pp. 78C-85C.
ISSN: 0002-9149.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Angiographic trials of coronary atherosclerosis treatment have demonstrated that lowering low density lipoprotein (LDL) cholesterol concentrations improves **coronary artery stenosis**. Most patients in previous trials have had at least mildly elevated LDL. Recently, however, the Harvard Atherosclerosis Reversibility Project (HARP) did not find such benefit in patients with lower baseline LDL levels compared with previous trials. We reviewed and analyzed all cholesterol-lowering trials that used angiographic endpoints. Unifactorial trials of hypocholesterolemic dietary or drug therapy demonstrated that the higher the baseline LDL, the greater the improvement in quantitatively determined stenosis in the treatment group compared with the controls (r = .83). Considering the change in stenosis in the treatment group alone, regression was more common in trials in which baseline mean LDL was gt 170 mg/dl (gt 4.4 mmol/liter), whereas progression occurred when baseline mean LDL was lt 170 mg/dl (lt 4.4 mmol/liter). HARP had the lowest baseline LDL (137 mg/dl (3.54 mmol/liter)), and showed no tendency for improvement in lesions. In contrast to the influence of baseline LDL levels, neither a low LDL level achieved on treatment nor a large percentage reduction in LDL was related to improvement in lesions. Sample size differences between HARP and the other trials are unlikely to be a major explanatory factor, since trials of comparable sample size to HARP, but with higher initial LDL, demonstrated favorable results. We conclude that coronary lesions that develop in the context of average LDL levels show less angiographic improvement in response to substantial LDL reduction than lesions in hypercholesterolemic patients. However, the clinical relevance of this finding awaits results from ongoing clinical endpoint trials in the normocholesterolemic population.

L5 ANSWER 2 OF 10 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 91146414 MEDLINE
DOCUMENT NUMBER: 91146414 PubMed ID: 1997308
TITLE: [Xanthomas of the Achilles tendon as the cardinal symptom of sitolsterolemia].
Xanthome der Achillessehnen als Leitsymptom der Sitosterinamie.
AUTHOR: Grahlke B K
CORPORATE SOURCE: Marinesanitatszentrum, Flensburg.
SOURCE: DEUTSCHE MEDIZINISCHE WOCHENSCHRIFT, (1991 Mar 1) 116 (9) 335-8.
Journal code: ECL; 0006723. ISSN: 0012-0472.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199104
ENTRY DATE: Entered STN: 19910419
Last Updated on STN: 19910419
Entered Medline: 19910403

AB The circumference of both Achilles tendons had gradually increased over the years in a now 32-year-old man (diameter of the left tendon 4.5 cm, of the right one 3.5 cm). This finally led to exercise-related pain in both tendons. Biopsy revealed benign deposition of xanthomata. Serum total cholesterol concentration was 261 mg/dl. Determination of various sitosterol fragments in serum gave a beta-sitosterol level of 43 mg/dl (normal range 0.3-1.7 mg/dl), characteristic of sitosterolaemia, which is an autosomal recessive disease causing intestinal hyperabsorption of a range of plant steroids closely related to cholesterol. On a **diet** low in plant steroids and treatment with cholestyramine (up to 32 g daily) the beta-sitosterol concentration fell, but only to 35 mg/dl, because of poor patient compliance. The patient died suddenly from **coronary artery stenosis** seven months after the diagnosis of sitosterolaemia.

L5 ANSWER 3 OF 10 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 92009631 MEDLINE
DOCUMENT NUMBER: 92009631 PubMed ID: 1916619
TITLE: [Modification of risk factors through physical training and low-fat **diet**].
Beeinflussung von Risikofaktoren durch korperliches training und fettarme Ernahrung.
AUTHOR: Schuler G; Hambrecht R; Schlierf G; Schneider J; Grunze M; Methfessel S; Hauer K; Kubler W
CORPORATE SOURCE: Medizinische Universitatsklinik Heidelberg.
SOURCE: HERZ, (1991 Aug) 16 (4) 237-42.
Journal code: F88; 7801231. ISSN: 0340-9937.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199111
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19920124
Entered Medline: 19911121

AB This intervention program investigated the applicability and the effects of intensive physical exercise and low-fat **diet** on the progression of coronary atherosclerotic lesions and stress induced myocardial ischemia in patients with stable angina pectoris. Patients participating in this study were recruited following routine coronary angiography for angina pectoris. Inclusion criteria were male sex, stable symptoms, a willingness to participate in the study for at least twelve months, and coronary artery stenoses well documented by angiography. Exclusion criteria were unstable angina pectoris, left main **coronary artery stenosis** greater than 25% luminal diameter reduction, severely depressed left ventricular ejection fraction (less than 35%), significant valvular heart disease, insulin-dependent diabetes mellitus, primary hypercholesterolemia (type II hyperlipoproteinemia, low-density lipoprotein greater than 210 mg/dl), and conditions precluding regular physical exercise. 18 patients participated in this program for one year; they consumed a low-fat, low-cholesterol **diet** (less than 20 energy % fat, cholesterol less than 200 mg/day) and exercised for more than 3 h/week. Myocardial oxygen consumption was estimated from maximum rate-pressure product at peak exercise; it was correlated to stress induced myocardial ischemia, as measured by 201Tl-scintigraphy. Results were compared with those of 18 matched patients on "usual care". In the intervention group, physical work

capacity (161 +/- 34 W vs. 194 +/- 42 W) and maximum rate pressure product (25.0 +/- 6.3 x 10(3) vs. 27.2 +/- 5.3 x 10(3)) increased significantly (p less than 0.01). Patients willing to devote time and effort to intensive physical exercise and to comply with a low-fat **diet** may benefit from this form of therapy.(ABSTRACT TRUNCATED AT 250 WORDS)

L5 ANSWER 4 OF 10 MEDLINE
ACCESSION NUMBER: 90165165 MEDLINE
DOCUMENT NUMBER: 90165165 PubMed ID: 2624364
TITLE: [Value of endomyocardial biopsy in congestive heart failure in diabetics without coronary disease]. Interet de la biopsie endomyocardique dans l'insuffisance cardiaque congestive du diabetique non coronarien.
AUTHOR: Valensi P; Sachs R N; Nitemberg A; Perennec-Cardinali J; Attali J R
CORPORATE SOURCE: Service d'Endocrinologie, Diabetologie, Nutrition, Hopital Jean-Verdier, Bondy.
SOURCE: ANNALES DE MEDECINE INTERNE, (1989) 140 (6) 473-6. Journal code: 5FZ; 0171744. ISSN: 0003-410X.
PUB. COUNTRY: France
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199003
ENTRY DATE: Entered STN: 19900601
Last Updated on STN: 19900601
Entered Medline: 19900328

AB Two cases of congestive heart failure in diabetic females are reported. One patient had moderate hypertension. Echography and angiography revealed a low-output dilated cardiopathy and ruled out the possibility of **coronary artery stenosis** and thin amyloid deposits were found in one patient. The evolution was favorable with a low-salt **diet**, associated with diuretic and vasodilator treatments. These case reports confirm the existence of a diabetic myocardiopathy, which may lead to congestive heart failure. They justify a complete hemodynamic analysis and a histopathological evaluation of the myocardium when this complication occurs.

L5 ANSWER 5 OF 10 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 89149869 MEDLINE
DOCUMENT NUMBER: 89149869 PubMed ID: 2920067
TITLE: Does platelet aggregation play a role in the reduction in localized intimal proliferation in normolipidemic pigs with fixed **coronary artery stenosis** fed dietary fish oil?.
AUTHOR: Hartog J M; Lamers J M; Essed C E; Schalkwijk W P; Verdouw P D
CORPORATE SOURCE: Laboratory for Experimental Cardiology, Erasmus University Rotterdam, The Netherlands.
SOURCE: ATHEROSCLEROSIS, (1989 Mar) 76 (1) 79-88. Journal code: 95X; 0242543. ISSN: 0021-9150.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198904
ENTRY DATE: Entered STN: 19900306
Last Updated on STN: 19980206
Entered Medline: 19890404

AB In order to investigate the effect of fish oil on intimal proliferation of coronary arteries with a fixed stenosis normolipidemic piglets received a basic **diet** to which either 9% (w/w) lard (L, n = 8) or 4.5%

(w/w) lard and 4.5% (w/w) mackerel oil (ML, n = 8) was added for 4 months. Stenosis was applied by implanting a 4.0 X 2.0 mm (i.d.) Teflon constrictor around the left anterior descending coronary artery (LADCA) (o.d. 2.7 +/- 0.1 mm). During the dietary period ADP-induced platelet aggregation in whole blood was higher in L than in ML. Partial replacement of 20:4 n - 6 by 20:5 n - 3 fatty acids in the platelet membranes of ML may have altered platelet aggregation by changes in eicosanoid synthesis. The plasma cholesterol and triglyceride levels did not change in L, but decreased in ML. At the end of the 4-month dietary period the animals were again anesthetized and regional myocardial perfusion (radioactive labelled microspheres) and systolic segment length shortening (SLS) were measured while the hearts were paced at 160 pulses/min. Perfusion and SLS of non-LADCA nourished segment were similar for L and ML. However, transmural flow to the LADCA perfused myocardium was impaired in both groups, but the deficiency in endocardial perfusion was considerably larger in L than in ML, resulting in a larger loss of SLS in the former. Remote (2-3 cm from the site of the constrictor) luminal encroachment was minimal (less than 2%) in both groups, but at the site of the constrictor there was significant encroachment in both groups which was higher in L (62 +/- 7%) than in ML (11 +/- 4%). It is thought that in these normolipidemic pigs the reduction in platelet aggregation may play a role in the smaller intimal proliferation of the fish oil-fed animals.

L5 ANSWER 6 OF 10 MEDLINE
ACCESSION NUMBER: 85267338 MEDLINE
DOCUMENT NUMBER: 85267338 PubMed ID: 3894896
TITLE: The place of coronary artery bypass surgery: an appraisal.
AUTHOR: Heller R F; Leeder S R
SOURCE: MEDICAL JOURNAL OF AUSTRALIA, (1985 Jul 22) 143 (2) 70-2.
Journal code: M26; 0400714. ISSN: 0025-729X.
PUB. COUNTRY: Australia
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198508
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850827

AB The enthusiasm for performing coronary artery bypass graft (CABG) surgery in Australia is increasing. The results of a number of careful trials which have compared surgical with medical treatment have now appeared. While there is agreement on both the increased survival provided by CABG surgery in those with left main **coronary artery stenosis** and the relief of symptoms in patients in whom medical therapy has failed to control severe angina, there is debate about the value of surgery in other types of disease. With improvements in medical therapy, the most recent trials have failed to show a significant overall survival benefit from surgery, although it is generally considered that surgery can relieve angina and that, in at least some groups of persons with stenosis of all three main coronary vessels (triple-vessel disease), surgery may prolong life. Alternative methods of prolonging survival among people with ischaemic heart disease include the reduction of risk factors (such as hypertension, raised blood cholesterol levels and cigarette smoking), as well as treating patients with beta-blocking agents after a myocardial infarction. We suggest it is likely that a combination of these approaches could be more effective in terms of lives saved than is CABG and may be less expensive. The current expansion of CABG surgery in Australia should be viewed in this light.

L5 ANSWER 7 OF 10 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 84052702 MEDLINE

DOCUMENT NUMBER: 84052702 PubMed ID: 6685520
TITLE: Effects of gender and social behavior on the development of coronary artery atherosclerosis in cynomolgus macaques.
AUTHOR: Hamm T E Jr; Kaplan J R; Clarkson T B; Bullock B C
CONTRACT NUMBER: HL-14164 (NHLBI)
R01 HL-26561 (NHLBI)
RR 07009 (NCRR)
+
SOURCE: ATHEROSCLEROSIS, (1983 Sep) 48 (3) 221-33.
Journal code: 95X; 0242543. ISSN: 0021-9150.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198312
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19831217

AB This experiment involved examination of the effects of gender and social status ('competitive dominance') on the coronary artery atherosclerosis of cynomolgus monkeys. Thirty-two adult Macaca fascicularis (16 males, 16 females) were fed a **diet** containing a moderate amount of cholesterol (0.56 mg/cal) for 16 months. The monkeys were housed in groups of 4 animals of the same sex, and all groups were stable in composition for the entire experiment. After 1 year a'competitive dominance' score was determined for each monkey, based on feeding order in 9 trials involving a preferred food as incentive. At necropsy the coronary arteries were pressure perfused; 5 sections each were then taken from the left anterior descending, left circumflex and right coronary arteries. For each animal, the mean percent lumen stenosis calculated from theses 15 sections was used as the index of extent of coronary artery atherosclerosis. Males had significantly more extensive coronary artery atherosclerosis than did females. Further, among both males and females, submissive animals (low in competitiveness) had more extensive **coronary artery stenosis** than did their dominant (highly competitive) counterparts. A similar pattern was observed in the thoracic and abdominal portions of the aorta with respect to competitiveness, but not gender. In the iliac artery, females had less atherosclerosis than males but there was no competitiveness effect. The gender and social status effects on atherosclerosis were each statistically independent of variability in clinical-pathological measures (serum lipid concentrations and heart weight). The results indicated that: (a) gender and psychosocial stress independently affect the development of coronary artery atherosclerosis; (b) the mechanisms mediating these effects remain unknown; and (c) the cynomolgus macaque is a good model for the study of such phenomena.

L5 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1982:272253 BIOSIS
DOCUMENT NUMBER: BA74:44733
TITLE: REDUCTION OF CORONARY ATHERO SCLEROSIS BY MODERATE CONDITIONING EXERCISE IN MONKEYS ON AN ATHEROGENIC **DIET**.
AUTHOR(S): KRAMSCH D M; ASPEN A J; ABRAMOWITZ B M; KREIMENDAHL T; HOOD W B JR
CORPORATE SOURCE: BOSTON UNIV. SCH. MED., 75 E. NEWTON ST., BOSTON, MA 02118.
SOURCE: N ENGL J MED, (1981) 305 (25), 1483-1489.
CODEN: NEJMAG. ISSN: 0028-4793.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB All available evidence that exercises may protect against coronary heart disease is circumstantial, and direct evidence is difficult to obtain in human beings. The effect of moderate conditioning with treadmill exercise

on developing coronary artery disease in monkeys (Macaca fascicularis) on an atherogenic **diet** was studied. Physical training was demonstrated by slow heart rates. Serum total cholesterol was the same (approximately 600 mg/dl or 15.5 mmol/l) in exercising and non-exercising monkeys, with significantly higher high density lipoprotein (HDL) cholesterol and much lower triglyceride and low density lipoprotein (LDL) plus very low density lipoprotein (VLDL) triglyceride in the exercise group. Ischemic ECG changes, angiographic signs of coronary artery narrowing, and sudden death were observed only in non-conditioned monkeys, in which post-mortem examination revealed marked coronary atherosclerosis and stenoses. Exercise was associated with substantially reduced overall atherosclerotic involvement, lesion size and collagen accumulation; it also produced much larger heart and wider coronary arteries, further reducing the degree of luminal narrowing. Moderate exercise may prevent or retard coronary heart disease in primates.

L5 ANSWER 9 OF 10 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1982:35706 BIOSIS
DOCUMENT NUMBER: BR22:35706
TITLE: REFINEMENT OF END POINT MEASUREMENT BASED ON A QUALITY CONTROL SUBSTUDY.
AUTHOR(S): DETRE K
CORPORATE SOURCE: DEP. OF EPIDEMIOL., UNIV. OF PITTSBURGH, PENNSYLVANIA.
SOURCE: COMBINED ANNUAL SCIENTIFIC SESSIONS OF THE SOCIETY FOR CLINICAL TRIALS AND THE 8TH ANNUAL SYMPOSIUM FOR COORDINATING CLINICAL TRIALS, SAN FRANCISCO, CALIF., USA, MAY 11-13, 1981. CONTROLLED CLIN TRIALS, (1981) 2 (1), 75. CODEN: CCLTDH.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L5 ANSWER 10 OF 10 MEDLINE
ACCESSION NUMBER: 81099607 MEDLINE
DOCUMENT NUMBER: 81099607 PubMed ID: 7454129
TITLE: Pathophysiology of long-chain polyene fatty acids in heart muscle.
AUTHOR: Gudbjarnason S
SOURCE: NUTRITION AND METABOLISM, (1980) 24 Suppl 1 142-6. Journal code: OAT; 0330472. ISSN: 0029-6678.
PUB. COUNTRY: Switzerland
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198103
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810324

AB The polyene fatty acid composition of cardiac phospholipids is modified by age, **diet** and stress in the rat. In the aging heart there is a progressive replacement of 18:2n6 by 20:4n6 in phosphatidylcholine (PC) and a replacement of 18:2n6 by 22:6n3 in phosphatidylethanolamine (PE). Norepinephrine stress accelerates aging of cardiac PC and PE. Dietary fish oil causes a replacement of 18:2n6 and 20:4n6 by 22:6n3 in cardiac PC and PE but not in cardiolipin. Studies on human cardiac autopsy samples suggest that: (a) polyene fatty acid composition changes with age; (b) stability of cardiac phospholipids is a function of the fatty acid composition, chain length and unsaturation; (c) coronary atherosclerosis is associated with a reduced content of 18:2n6 in phospholipids, an increased content of glycerides of abnormal composition and an unexpectedly low level of free fatty acids (FFA) in the heart muscle, and (d) many cases of sudden cardiac death in the absence of marked

coronary artery stenosis or myocardial
infarction may be associated with significant alterations in myocardial
levels of FFA (increase) or PE (decrease).

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     (FILE 'HOME' ENTERED AT 17:15:24 ON 16 JAN 2002)

     FILE 'MEDLINE, BIOSIS' ENTERED AT 17:15:37 ON 16 JAN 2002
L1          3785 S CORONARY(W)ARTERY(W)STENOSIS
L2             5 S L1 AND FISH(W)OIL?
L3             4 DUP REM L2 (1 DUPLICATE REMOVED)
L4            14 S L1 AND (DIET OR (LOWER? OR REDUC?)(W)(CHOLESTEROL OR FAT)(W)I
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Question + Diet

1: Circulation 1984 Feb;69(2):313-24

Effects of therapy with cholestyramine on progression of coronary arteriosclerosis: results of the NHLBI Type II Coronary Intervention Study.

Brensike JF, Levy RI, Kelsey SF, Passamani ER, Richardson JM, Loh IK, Stone NJ, Aldrich RF, Battaglini JW, Moriarty DJ, et al.

In the National Heart, Lung and Blood Institute Type II Coronary Intervention Study, patients with Type II hyperlipoproteinemia and coronary artery disease (CAD) were placed on a low-fat, low-cholesterol diet and then were randomly allocated to receive either 6 g cholestyramine four times daily or placebo. This double-blind study evaluated the effects of cholestyramine on the progression of CAD as assessed by angiography. Diet alone reduced the low-density lipoprotein cholesterol 6% in both groups. After randomization, low-density lipoprotein cholesterol decreased another 5% in the placebo group and 26% in the cholestyramine-treated group. Coronary angiography was performed in 116 patients before and after 5 years of treatment. CAD progressed in 49% (28 of 57) of the placebo-treated patients vs 32% (19 of 59) of the cholestyramine-treated patients (p less than .05). When only definite progression was considered, 35% (20 of 57) of the placebo-treated patients vs 25% (15 of 59) of the cholestyramine-treated patients exhibited definite progression; the difference was not statistically significant. However, when this analysis was performed with adjustment for baseline inequalities of risk factors, effect of treatment was more pronounced. Of lesions causing 50% or greater stenosis at baseline, 33% of placebo-treated and 12% of cholestyramine-treated patients manifested lesion progression (p less than .05). Similar analyses with other end points (percent of baseline lesions that progressed, lesions that progressed to occlusion, lesions that regressed, size of lesion change, and all cardiovascular end points) all favored the cholestyramine-treated group, but were not statistically significant. Thus, although the sample size does not allow a definitive conclusion to be drawn, this study suggests that cholestyramine treatment retards the rate of progression of CAD in patients with Type II hyperlipoproteinemia.

Publication Types:
- Clinical Trial
- Randomized Controlled Trial

Order 1-17-02

Fish oil + Eicosa. & Docosahex.

L20 ANSWER 170 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1992:393232 BIOSIS
DOCUMENT NUMBER: BA94:65407
TITLE: THE EFFECT OF FISH FATS ON THE COMPOSITION OF FATTY ACIDS IN TISSUE LIPIDS DURING EXPERIMENTAL HYPERCHOLESTEROLAEMIA.
AUTHOR(S): ZIEMLANSKI S; BUDZYNSKA-TOPOLOWSKA J; RODKIEWICZ B; KOLAKOWSKA A
CORPORATE SOURCE: UL. BARSKA 5 M. 19, 02-315 WARSZAWA, POL.
SOURCE: ZYWIENIE CZLOWIEKA METAB, (1992) 19 (2), 71-85.
CODEN: ZCMEDQ. ISSN: 0209-164X.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB The effect of **fish oil** on the composition of fatty acids in serum lipids and in certain tissues (reserve fatty tissue, liver, heart, testicles) was studied in guinea pigs during experimental hypercholesterolaemia. During 12 weeks the animals were kept on a diet with 14% of kcal derived from the studied fat and 0.1% **cholesterol** . Two control groups recieved granulated animal food without **cholesterol** or with 0.1% **cholesterol**. No strict correlation was found between the content of **eicosapentaenoic** acid and **docosahexaenoic** acid in myocardial lipids and their content in the diets - in liver lipids the content of **docosahexaenoic** acid was higher than in the diets. The addition of **cholesterol** to the diet disturbed the metabolism of unsaturated fatty acids from sthe n-6 group and n-3 group in myocardial lipids. In all animals with hypercholesterolaemia the testicular lipids contained higher amounts of polyunsaturated fatty acids, especially arachidonic acid with low amounts of this acid and its precusor linolic acid in the diets.

L20 ANSWER 171 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1992:114606 BIOSIS
DOCUMENT NUMBER: BA93:60406
TITLE: DIETARY EFFECTS OF OMEGA-3 POLYUNSATURATED FATTY ACIDS ON BODY FAT COMPOSITION AND HEALTH STATUS OF FARM-RAISED BLUE AND SILVER FOXES.
AUTHOR(S): ROUVINEN K
CORPORATE SOURCE: NOVA SCOTIA AGRIC. COLL., DEP. ANIMAL SCI., P.O. BOX 550, TRURO, NOVA SCOTIA, B2N 5E3, CAN.
SOURCE: ACTA AGRIC SCAND, (1991) 41 (4), 401-414.
CODEN: AASCAU. ISSN: 0001-5121.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Farm-raised blue and silver foxes were fed diets based on slaughterhouse offal (SH) and fish mixture supplemented with **fish oil** (FM) from weaning to pelting in order to clarify the effects of accumulation of omega-3 fatty acids in the tissues and organs. Some blue foxes were also fed an antioxidant (Rexoquin, 200-1000 ppm) supplemented diet. The dietary background of the animals significantly influenced the fatty acid composition of all body fat depots in both fox species. The animals of the FM group had considerably more **eicosapentaenoic** (EPA), **docosahexaenoic** (DHA) and cetoleic acids in their tissues than the animals of the SH group. In silver fox livers, the amount of DHA was even higher than in blue foxes. Fat accumulation pattern of the blue and silver fox livers also differed considerably between the diets. In the SH diets fat accumulated in the liver in large droplets, while in the FM diets it was present in small droplets. Furthermore, degenerative changes were more numerous and severe in the FM dietary group. The antioxidant supplementation of the blue fox diets employed appeared to be toxic to the animals. It increased liver fat content, which was also seen as fatty degeneration of the liver. The increase in the levels of the serum transaminases ALAT and ASAT was clearly connected with the disturbances in

liver functions and degenerative changes. Also an increase in serum **cholesterol** was observed in animals with cholestasis. Liver vitamin A and selenium levels were higher in the FM diets in silver foxes. In blue foxes, the antioxidant supplementation employed had no influence on the vitamin status of the animals.

L20 ANSWER 172 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:504878 BIOSIS
DOCUMENT NUMBER: BA92:127838
TITLE: ANALYSIS OF THE FATTY ACID COMPOSITION OF THE LIPID CLASSES IN HUMAN BLOOD SERUM UNDER NORMAL DIET AND WHEN SUPPLEMENTED WITH **FISH OIL**.
AUTHOR(S): LIEBICH H M; JAKOBER B; WIRTH C; PUKROP A; EGGSTEIN M
CORPORATE SOURCE: MEDIZINISCHE UNIVERSITAETSKLINIK, D-7400 TUEBINGEN, GERMANY.
SOURCE: HRC (J HIGH RESOLUT CHROMATOGR), (1991) 14 (7), 433-437. CODEN: JHRCE7. ISSN: 0935-6304.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Total lipids have been extracted from human serum with chloroform-methanol 2:1 (v/v) and separated into individual classes by TLC. After transesterification the fatty acid methyl esters were analyzed by capillary gas chromatography on an FFAP column. The quantitation of .omega.-3 fatty acids has been performed using internal and external standards. Internal lipid standards for each lipid class were carried throughout the entire analytical procedure. Under normal diet **eicosapentaenoic** acid (EPA) and **docosahexaenoic** acid (DHA) are incorporated into the lipid classes to different extents: **cholesterol** esters; EPA, 6.5 .+-. 1.9 .gamma./ml serum; DHA, 4.3 .+-. 1.9 .mu.g/ml; phospholipids; EPA, 5.9 .+-. 2.7 .mu.g/ml; DHA, 31.8 .+-. 8.1 .mu.g/ml. **Fish oil** supplementation leads to a 4 to 6-fold rise in EPA and to an approximately 2-fold rise in DHA.

L20 ANSWER 173 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:470787 BIOSIS
DOCUMENT NUMBER: BR41:96547
TITLE: EFFECT OF **FISH-OIL** ON TRANSSPLANCHNIC LIPID BALANCE AND TRANSFEMORAL PLASMA FLOW IN HEALTHY MAN.
AUTHOR(S): WALDHAUSL W; RATHEISER K; GASIC S; HUTTINGER C; NOWOTNY P; VIERHAPPER H
CORPORATE SOURCE: I. MED. UNIV., DIV. CLINICAL ENDOCRINOL. DIABETOL., VIENNA, AUSTRIA.
SOURCE: 25TH MEETING OF THE EUROPEAN SOCIETY FOR CLINICAL INVESTIGATION, PISA, ITALY, APRIL 3-6, 1991. EUR J CLIN INVEST, (1991) 21 (2 PART 2), 35. CODEN: EJCIB8. ISSN: 0014-2972.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L20 ANSWER 174 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:367819 BIOSIS
DOCUMENT NUMBER: BA92:56044
TITLE: THE EFFECT OF SHORT-TERM OMEGA-3 POLYUNSATURATED FATTY ACID SUPPLEMENTATION IN PATIENTS WITH CHRONIC RENAL INSUFFICIENCY.
AUTHOR(S): SCHAAP G H; BILO H J G; BEUKHOF J R; GANS R O B; POPP-SNIJDERS C; DONKER A J M
CORPORATE SOURCE: DIVISION NEPHROLOGY, DEP. INTERNAL MED., UNIV. HOSP., P.O. BOX 9101, 6500 HB NIJMEGEN, THE NETH.
SOURCE: CURR THER RES, (1991) 49 (6), 1061-1070. CODEN: CTCEA9. ISSN: 0011-393X.

FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Previous studies in patients with moderate to end-stage renal insufficiency revealed varying results of omega-3 polyunsaturated fatty acids (omega-3 PUFA) on renal function, viscosity of erythrocyte suspensions, and lipid profile. In order to further elucidate the influence of omega-3 PUFA on these variables, seven non-diabetic patients with chronic renal insufficiency ingested six capsules of **fish oil** (containing 1,800 mg of **eicosapentaenoic** acid C20:5 omega-3 and 1,200 mg of **docosahexaenoic** acid C22:6 omega-3) daily for 12 weeks. Measurements were performed at baseline, after 12 weeks of **fish oil** ingestion, and again 12 weeks after withdrawal of the supplementation. Glomerular filtration rate and effective renal plasma flow did not change in this short-term study. Erythrocyte viscosity, measured at hematocrit of 0.80 in buffer with a Contraves low shear 30 rheometer at various shear rates, improved significantly, especially at the lower shear rates. Lipid profiles analysis (ultracentrifuge technique) showed a significant rise in high-density lipoprotein (HDL)2- and total HDL-**cholesterol**, and a decrease in very low-density lipoprotein-**cholesterol** and triglycerides concentration. However, low-density lipoprotein (LDL)-**cholesterol** rose as well and total **cholesterol** tended to rise. HDL2/LDL-ratio and HDL/LDL-ratio did not change. We conclude that daily addition of small amounts of omega-3 PUA to the diet of patients with moderate to severe renal insufficiency has, in addition to a beneficial effect on erythrocyte deformability, both positive and negative effects on lipid profile. Whether the favorable effects outweigh the unfavorable effects remains to be clarified.

L20 ANSWER 175 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:331336 BIOSIS
DOCUMENT NUMBER: BR41:27886
TITLE: EFFICACY FOR LOWERING PLASMA LIPID LEVELS COMPARISON OF SINGLE FATTY ACIDS WITH WHOLE **FISH OIL**.
AUTHOR(S): OH S Y; FESSLER T A
CORPORATE SOURCE: DEP. DIETETICS AND NUTRITION, UNIV. KANS. MED. CENT., KANSAS CITY, KANS. 66103.
SOURCE: 75TH ANNUAL MEETING OF THE FEDERATION OF AMERICAN SOCIETIES FOR EXPERIMENTAL BIOLOGY, ATLANTA, GEORGIA, USA, APRIL 21-25, 1991. FASEB (FED AM SOC EXP BIOL) J, (1991) 5 (6), A1640.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L20 ANSWER 176 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:63837 BIOSIS
DOCUMENT NUMBER: BR40:29192
TITLE: N-3 POLYUNSATURATED FATTY ACIDS DECREASE SATURATION BUT DO NOT PREVENT PRECIPITATION OF **CHOLESTEROL** CRYSTALS IN BILE.
AUTHOR(S): BERR F; HOLL J; FISCHER S; RICHTER O W; MAYER M; PAUMGARTNER G
CORPORATE SOURCE: DEP. MED. II, UNIV. MUNICH, FRG.
SOURCE: 41ST ANNUAL MEETING OF THE AMERICAN ASSOCIATION FOR THE STUDY OF LIVER DISEASES, CHICAGO, ILLINOIS, USA, NOVEMBER 3-6, 1990. HEPATOLOGY, (1990) 12 (4 PART 2), 898.
CODEN: HPTLD9. ISSN: 0270-9139.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L20 ANSWER 177 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:39196 BIOSIS
DOCUMENT NUMBER: BR40:16176
TITLE: DIFFERING EFFECTS OF FISH AND **FISH OIL** IN HYPERLIPIDEMIC MEN.
AUTHOR(S): CLIFTON P M; COBIAC L; ABBEY M; NESTEL P J
CORPORATE SOURCE: CSIRO DIV. OF HUMAN NUTRITION, ADELAIDE, AUST.
SOURCE: 63RD SCIENTIFIC SESSIONS OF THE AMERICAN HEART ASSOCIATION, DALLAS, TEXAS, USA, NOVEMBER 12-15, 1990. CIRCULATION, (1990) 82 (4 SUPPL 3), III476.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L20 ANSWER 178 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:13745 BIOSIS
DOCUMENT NUMBER: BR40:2075
TITLE: NUTRITION OF RHEUMATOID ARTHRITIS SHOULD IT BE CONSIDERED A SUPPLEMENTAL TREATMENT.
AUTHOR(S): OJEDA S; MARTIN E
CORPORATE SOURCE: SERV. DE REUMATOLOGIA, HOSP. LA PAZ, MADRID, SPAIN.
SOURCE: Rev. Esp. Reumatol., (1990) 17 (3), 110-114.
CODEN: RERMAW.
FILE SEGMENT: BR; OLD
LANGUAGE: Spanish

L20 ANSWER 179 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:7771 BIOSIS
DOCUMENT NUMBER: BA91:7771
TITLE: INVESTIGATION OF THE MECHANISMS OF INCREASE IN SERUM HIGH DENSITY LIPOPROTEIN **CHOLESTEROL** BY CONCENTRATED **FISH OIL** IN RATS.
AUTHOR(S): MU Z; LIU Y; SUN M; ZHANG S
CORPORATE SOURCE: INST. HYGIENE ENVIRON. MED. ACAD. MILITARY, MED. SCI, TIANJIN 300050.
SOURCE: ACTA NUTR SIN, (1990) 12 (2), 134-138.
CODEN: YYHPA4. ISSN: 0512-7955.
FILE SEGMENT: BA; OLD
LANGUAGE: Chinese
AB In this experiment, we investigate with enzymological methods the mechanisms of increase in serum high density lipoprotein **cholesterol** (HDL-C) in rats fed with concentrated **fish oil**. The rats were fed with high fat diet (Group 1), high fat plus olive oil (Group 2) and high fat plus concentrated **fish oil** (Group 3) for 6 weeks respectively. The concentrated **fish oil** contained about 26% methyl-**eicosapentaenoate** (EPA-M) and 52% methyl-**docosahexaenoate** (DHA-M), and was given in 0.5 ml/day for each animal. The results showed that HDL-C levels in the serum of rats fed with **fish oil** were markedly higher than Group 1 and 2 ($p < 0.01$). The elevation of HDL-C was due to the increase of subgroup 2 of HDL-C, while subgroup 3 of HDL-C did not change obviously. The activities of serum lecithin **cholesterol** acyltransferase (LCAT) and lipoprotein lipase (LPL) were significantly higher ($p < 0.01$) but the activity of hepatic endothelial lipase (HEL) was significantly low ($p < 0.05$) in Group 3 as compared with Group 1 and 2. The present study demonstrated that **fish oil** could elevate HDL-C level through the following mechanisms: increasing activities of LCAT and LPL; inhibiting the activity of HEL.

L20 ANSWER 180 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1990:217452 BIOSIS
DOCUMENT NUMBER: BA89:114742
TITLE: SIMPLE HIGH VACUUM DISTILLATION EQUIPMENT FOR DEODORIZING **FISH OIL** FOR HUMAN CONSUMPTION.
AUTHOR(S): DINAMARCA E; GARRIDO F; VALENZUELA A
CORPORATE SOURCE: INST. NUTR. TECNOLOGIA ALIMENTOS, UNIV. CHILE, CASILLA 138-11, SANTIAGO 11, CHILE.
SOURCE: LIPIDS, (1990) 25 (3), 170-171.
CODEN: LPDSAP. ISSN: 0024-4201.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB A simple piece of glass equipment for deodorizing **fish oil** (sardine oil) by high vacuum distillation was designed and constructed. The equipment has a throughput of 450-500 ml/hr working at 140.degree. C and at a constant pressure of 2 .times. 10-2 mm Hg. It reduces the peroxide value and the **cholesterol** content of the oil and improves the flavor without affecting the EPA and DHA content.

L20 ANSWER 181 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1989:529367 BIOSIS
DOCUMENT NUMBER: BR37:128225
TITLE: METABOLIC EFFECTS OF **FISH-OIL** IN SUBJECTS WITHOUT AND WITH IMPAIRED GLUCOSE TOLERANCE.
AUTHOR(S): RATHEISER K; WALDHAUSL W; KOMJATI M; FASCHING P; OSTERODE W; ROHAC M; VIERHAPPER H
CORPORATE SOURCE: 1ST DEP. INTERNAL MED., VIENNA, AUSTRIA.
SOURCE: 25TH ANNUAL MEETING OF THE EUROPEAN ASSOCIATION FOR THE STUDY OF DIABETES, LISBON, PORTUGAL, SEPTEMBER 20-23, 1989. DIABETOLOGIA, (1989) 32 (7), 532A.
CODEN: DBTGAJ. ISSN: 0012-186X.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L20 ANSWER 182 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1989:487298 BIOSIS
DOCUMENT NUMBER: BR37:108417
TITLE: **FISH OIL** REDUCES **CHOLESTEROL** AND ARACHIDONIC ACID CONTENT MORE EFFICIENTLY IN RATS FED DIETS CONTAINING LOW LINOLEIC ACID TO SATURATED FATTY ACID RATIO.
AUTHOR(S): GARG M L; WIERZBICKI A A; THOMSON A B R; CLANDININ M T
CORPORATE SOURCE: UNIV. ALBERTA, EDMONTON, CAN.
SOURCE: ANNUAL MEETING OF THE SOCIETE CANADIENNE DE RECHERCHES CLINIQUES (CANADIAN SOCIETY FOR CLINICAL INVESTIGATION), EDMONTON, ALBERTA, CANADA, SEPTEMBER 22-25, 1989. CLIN INVEST MED, (1989) 12 (SUPPL 4), B62.
CODEN: CNVMDL. ISSN: 0147-958X.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L20 ANSWER 183 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1989:487297 BIOSIS
DOCUMENT NUMBER: BR37:108416
TITLE: DIETARY **CHOLESTEROL** AND-OR OMEGA-3 FATTY ACID MODULATE DELTA-9 DESATURASE ACTIVITY IN RAT LIVER MICROSOMES.
AUTHOR(S): GARG M L; WIERZBICKI A A; THOMSON A B R; CLANDININ M T
CORPORATE SOURCE: UNIV. ALBERTA, EDMONTON, CAN.
SOURCE: ANNUAL MEETING OF THE SOCIETE CANADIENNE DE RECHERCHES

CLINIQUES (CANADIAN SOCIETY FOR CLINICAL INVESTIGATION), EDMONTON, ALBERTA, CANADA, SEPTEMBER 22-25, 1989. CLIN INVEST MED, (1989) 12 (SUPPL 4), B62.
CODEN: CNVMDL. ISSN: 0147-958X.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L20 ANSWER 184 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1989:157105 BIOSIS
DOCUMENT NUMBER: BA87:79206
TITLE: TRIGLYCERIDE LOWERING IN NEPHROTIC SYNDROME PATIENTS CONSUMING A **FISH OIL** CONCENTRATE.
AUTHOR(S): BAKKER D J; HABERSTROH B N; PHILBRICK D J; HOLUB B J
CORPORATE SOURCE: DEP. NUTR. SCI., COLL. BIOL. SCI., UNIV. GUELPH, GUELPH, ONT. N1G 2W1, CAN.
SOURCE: NUTR RES, (1989) 9 (1), 27-34.
CODEN: NTRSDC. ISSN: 0271-5317.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Alterations in serum lipids including a significant decrease in triglyceride with or without a reduction in total **cholesterol** and increase of high-density lipoprotein (HDL) **cholesterol** after dietary **fish oil** supplementation have been documented in recent literature. An attempt has been made to examine the effect of the n-3 polyunsaturated fatty acids as **eicosapentaenoic** acid (EPA, 20:5n-3) plus **docosahexaenoic** acid (DHA, 22:6n-3) on the serum lipids of human nephrotic syndrome patients. This patient group is typically hyperlipidemic, placing the patient at high atherosclerotic risk. In the present study, 9 nephrotic syndrome patients received an encapsulated fish lipid concentrate (MaxEPA) for 9 days. The level of serum triglyceride, total **cholesterol**, and HDL-**cholesterol** were measured at day 0, after 9 days of treatment, and 9 days after supplementation ceased. A significant decrease in serum triglyceride (by 31%) was observed, while there were no overall changes in the total **cholesterol** and HDL-**cholesterol** levels. Our results suggest that dietary **fish oil** supplementation may possibly offer benefit to some nephrotic syndrome patients since triglyceride-lowering in considered to have a protective effect against the development of cardiovascular disease.

L20 ANSWER 185 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1989:31385 BIOSIS
DOCUMENT NUMBER: BA87:19385
TITLE: SEMI-PREPARATIVE HPLC FRACTIONATION OF CONSUMER **FISH OIL** TRIACYLGLYCEROLS.
AUTHOR(S): WOJTUSIK M J; BROWN P R; TURCOTTE J G
CORPORATE SOURCE: DEP. CHEM., UNIV. R.I., KINGSTON, R.I. 02881.
SOURCE: J LIQ CHROMATOGR, (1988) 11 (9-10), 2091-2108.
CODEN: JLCHD8. ISSN: 0148-3919.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB A reversed-phase high performance liquid chromatographic method was developed for the semi-preparative fractionation of **fish oil**-derived triacylglycerols containing the esterified omega-3 fatty acids all-cis-5,8,11,14,17-**eicosapentaenoic** acid [EPA] and all-cis-4,7,10,13,17,20-**docosahexaenoic** acid [DHA]. Analytical separation conditions, such as mobile phase composition and flow rate could be directly applied to the semi-preparative mode, which was further optimized. Separation of triacylglycerol fractions was obtained in 15 minutes using flow rates of 3.0 ml/min with a mobile phase of acetone/acetonitrile (65:35, v/v). 250-mg samples of the **fish**

oil were fractionated and multi-milligram quantities of triacylglycerols were separated, which were 65% enriched in esterified EPA and DHA; a production rate of 500 mg/hr of this enriched fraction was obtained.

L20 ANSWER 186 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1988:379598 BIOSIS
DOCUMENT NUMBER: BA86:63508
TITLE: OMEGA-3 FATTY ACID LEVELS AND PERFORMANCE OF BROILER CHICKENS FED REDFISH MEAL OR REDFISH OIL.
AUTHOR(S): HULAN H W; ACKMAN R G; RATNAYAKE W M N; PROUDFOOT F G
CORPORATE SOURCE: RES. STN., AGRIC. CANADA, KENTVILLE, NOVA SCOTIA, CANADA B4N 1J5.
SOURCE: CAN J ANIM SCI, (1988) 68 (2), 533-548.
CODEN: CNJNAT. ISSN: 0008-3984.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB Arbor Acre broiler chickens were fed six different diets to determine if the omega-3 fatty acid content of broiler chicken carcasses could be enchanced by feeding additional redfish meal (RFM) or redfish oil (RFO). The six diets were: control (no fish meal or **fish oil**); 7.5% RFM, 15.0% RFM, 30.0% RFM, 2.1% RFO and 4.2% RFO. Mortality at 28 d and 42 d was lower ($P < 0.05$) for birds fed RFO compared to those fed RFM. Feeding additional RFM or RFO had no ($P > 0.05$) effect on mortality, but resulted in lower body weights ($P < 0.01$) and feed consumption ($P < 0.05$) and poorer ($P < 0.05$) feed conversion. Additions of RFM or RFO to the diets resulted in a substantial dietary enrichment of omega-3 fatty acids (especially **eicosapentaenoic** acid, EPA or 20:5n-3; and **docosahexaenoic** acid, DHA or 22:6n-3). Analyses (wt/wt%) revealed that breast meat was lower ($P < 0.001$) in lipid and triglyceride but higher in **cholesterol** esters ($P < 0.005$), free **cholesterol** ($P < 0.001$) and phospholipid ($P < 0.001$) than thigh meat. Lipid, free cholestrol and phospholipid of edible meat lipid increased with duration of feeding (14 d, 28 d, 42 d) but triglyceride content decreased. Dietary treatment had no effect ($P > 0.05$) on carcass lipid content or composition. Breast meat lipid contained more ($P < 0.001$) of the omega-3 fatty acids (especially EPA and DHA), more n-3 docosapentaenoic acid (DPA or 22:5n-3) and more total n-3 polyunsaturated fatty acid (n-3 PUFA) than thigh meat lipids. EPA, DPA, DHA and total n-3 PUFA in edible meat lipids increased ($P < 0.05$) with duration of feeding. Feeding additional RFM and RFO resulted in an increased accumulation of the EPA ($P < 0.001$), DPA ($P < 0.01$), DHA ($P < 0.01$) and total n-3 PUFA ($P < 0.001$), primarily at the expense of the omega-6 fatty acids linoleic (18:2n-6) and arachidonic (20:4n-6). It can be calculated from the data presented that on average a normal meal (100 g) of chicken which has been fed 7.5% fish meal, would contribute 140 mg of omega-3 fatty acids (EPA + DPA + DHA). The same size meal of cod flesh would contribute about 135 mg of these fatty acids.

L20 ANSWER 187 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1988:193193 BIOSIS
DOCUMENT NUMBER: BR34:96380
TITLE: EFFECT OF LOW MAINTENANCE DOSE OMEGA 3 FATTY ACIDS ON SERUM LIPID CONCENTRATIONS.
AUTHOR(S): ISMAIL S; BRANNIGAN M; O'CALLAGHAN D; HORGAN J H
CORPORATE SOURCE: DEP. CARDIOL., ST. LAURENCE'S HOSP., DUBLIN, IREL.
SOURCE: AUTUMN MEETING OF THE BRITISH CARDIAC SOCIETY, LONDON, ENGLAND, UK, NOVEMBER 24-26, 1987. BR HEART J, (1988) 59 (1), 126-127.
CODEN: BHJUAV. ISSN: 0007-0769.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD

LANGUAGE: English

L20 ANSWER 188 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1988:136425 BIOSIS
DOCUMENT NUMBER: BA85:71252
TITLE: RESPONSE OF PLASMA AND LIVER **CHOLESTEROL** AND FATTY ACIDS IN HYPERCHOLESTEROLEMIC RATS TO SHORT-TERM FEEDING OF VEGETABLE AND FISH OILS.
AUTHOR(S): HUANG Y-S; MCADOO K R; HORROBIN D F
CORPORATE SOURCE: EFAMOL RES. INST., KENTVILLE, N.S., CAN. B4N 4H8.
SOURCE: NUTR REP INT, (1987) 36 (6), 1171-1184.
CODEN: NURIBL. ISSN: 0029-6635.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB The changes of plasma and liver **cholesterol** contents and fatty acid compositions in hypercholesterolemic rats in response to 4-day feeding of either n-6 fatty acid-rich safflower oil or n-3 fatty acid-rich **fish oil** were examined. Results show that both dietary fats were equally effective in lowering plasma and liver **cholesterol**. In plasma and liver phospholipids, the n-6 acids in safflower oil-fed rats, and the n-3 acids in **fish oil** -fed rats were metabolized in a similar pattern: they rose rapidly on the first day of feeding, and increased less rapidly thereafter. The n-3 and n-6 polyunsaturated fatty acids in plasma cholesteryl esters were metabolized differently. In animals fed safflower oil, the proportions of 18:2n-6 were elevated rapidly after one day on the diet, and remained constant thereafter, while the levels of 20:4n-6 increased after the second day. In animals fed **fish oil**, the levels of 20:5n-3 increased steadily throughout the feeding, while those of 22:6n-3 increased only marginally. The implications of these results for the mechanism of the hypocholesterolemic effects of n-3 and n-6 fatty acids are discussed.

L20 ANSWER 189 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1987:306174 BIOSIS
DOCUMENT NUMBER: BR33:27847
TITLE: EFFECTS OF **FISH OIL** FO ON SERUM LIPIDS IN COLLEGE MEN IN A CONTROLLED FEEDING TRIAL.
AUTHOR(S): DELANY J; SNOOK J; ANDERSON P; VIVIAN V
CORPORATE SOURCE: OHIO STATE UNIV., COLUMBUS, OHIO 43210.
SOURCE: 71ST ANNUAL MEETING OF THE FEDERATION OF AMERICAN SOCIETIES FOR EXPERIMENTAL BIOLOGY, WASHINGTON, D.C., USA, MARCH 29-APRIL 2, 1987. FED PROC, (1987) 46 (4), 1172.
CODEN: FEPRA7. ISSN: 0014-9446.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L20 ANSWER 190 OF 190 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1983:332230 BIOSIS
DOCUMENT NUMBER: BA76:89722
TITLE: THE INFLUENCE OF DIFFERENT TYPES OF OMEGA-3 POLY UNSATURATED FATTY-ACIDS ON BLOOD LIPIDS AND PLATELET FUNCTION IN HEALTHY VOLUNTEERS.
AUTHOR(S): SANDERS T A B; ROSHANAI F
CORPORATE SOURCE: DEP. NUTRITION, QUEEN ELIZABETH COLL., UNIV. LONDON, CAMPDEN HILL ROAD, LONDON W8 7AH, U.K.
SOURCE: CLIN SCI (LOND), (1983) 64 (1), 91-100.
CODEN: CSCIAE. ISSN: 0143-5221.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Five healthy [human] subjects took a daily supplement of 20 ml of linseed

oil for 2 wk. After a break of at least 6 wk, the same subjects took a similar amount of MaxEPA (a **fish oil** fraction) for 2 wk. The linseed oil supplement provided 9.38 g of linolenic acid (18:3 .omega.3) and the MaxEPA supplement provided 3.03 g of **eicosapentaenoic** acid (20:5 .omega.3) and 2.93 g of **docosahexaenoic** acid (22:6 .omega.3). The effects of the supplements on plasma lipid concentrations and on the fatty acid composition of platelet phosphoglycerides were studied. In a 2nd experiment, 5 male subjects took 5, 10 and 20 g of MaxEPA/day in random order for 3 wk periods; each experimental period was separated by a break of at least 6 wk. These doses of MaxEPA provided 0.83, 1.67 and 3.33 g of 20:5 .omega.3 and 0.80, 1.61 and 3.22 g 22:6 .omega.3, respectively. The effects of these supplements on plasma lipid concentrations, the fatty acid composition of platelet phosphoglycerides, template bleeding time and platelet aggregation induced by collagen and the prostaglandin analogue compound U46619 [((15S)-hydroxy-11.alpha.,9.alpha.-epoxymethano)prosta-5Z,13E dienoic acid] were studied. In the platelet lipids, the proportion of 20:5 .omega.3 was increased by the 20 ml linseed oil supplement but the increase was small compared with the increase brought about by even 5 g of MaxEPA/day. The proportion of arachidonic acid (20:4 .omega.3) was substantially decreased by the MaxEPA supplement but not by the linseed oil supplement. The ratio of 20:4 .omega.6/20:5 .omega.3 fell from 32:1 in the control periods of 11.1 with 5 g, 7:1 with 10 g and 5:1 with 20 g of MaxEPA/day. The MaxEPA supplement also led to increases in the proportions of 22:5 .omega.3 and 22:6 .omega.3 and decreases in those of 20:3 .omega.6 and 22:4 .omega.6. Bleeding times tended to be prolonged with the MaxEPA supplement but did not follow any dose-dependent trend. Platelet aggregation induced by both collagen and compound U46619 was not inhibited in vitro. Plasma triglyceride concentrations were lowered by the MaxEPA supplement but not by the linseed oil supplement. Plasma triglyceride concentrations were substantially lowered by 10 g and 20 g of MaxEPA/day. Total plasma **cholesterol** concentrations were slightly lowered and HDL [high density lipoprotein] **cholesterol** concentrations were slightly increased by 20 g of MaxEPA/day. No other significant differences were noted. [The association between dietary fish derived fats and a lower incidence of ischemic heart disease is discussed.]

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     (FILE 'HOME' ENTERED AT 17:15:24 ON 16 JAN 2002)

     FILE 'MEDLINE, BIOSIS' ENTERED AT 17:15:37 ON 16 JAN 2002
L1          3785 S CORONARY(W)ARTERY(W)STENOSIS
L2             5 S L1 AND FISH(W)OIL?
L3             4 DUP REM L2 (1 DUPLICATE REMOVED)
L4            14 S L1 AND (DIET OR (LOWER? OR REDUC?)(W)(CHOLESTEROL OR FAT)(W)I
L5            10 DUP REM L4 (4 DUPLICATES REMOVED)
L6             2 S L1 AND NIACIN
L7             2 DUP REM L6 (0 DUPLICATES REMOVED)
L8            46 S L1 AND ASPIRIN
L9            34 DUP REM L8 (12 DUPLICATES REMOVED)
L10            4 S L1 AND (COLESTIPOL OR COLESTID OR QUESTRAN OR CHOLESTYRAMINE
L11            3 DUP REM L10 (1 DUPLICATE REMOVED)

     FILE 'REGISTRY' ENTERED AT 17:27:01 ON 16 JAN 2002
                  E BUSPIRONE
                  E BUSPIRONE/CN
L12            1 S E3

     FILE 'MEDLINE, BIOSIS' ENTERED AT 17:27:30 ON 16 JAN 2002
L13            0 S L1 AND L12
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L14	1	S L1 AND (EICOSAPENT? OR DOCOHEXA?)
L15	2023	S (EICOSAPENT? OR DOCOHEXA?) AND FISH(W)OIL
L16	3	S (EICOSAPENT? AND DOCOHEXA?) AND FISH(W)OIL
L17	2	DUP REM L16 (1 DUPLICATE REMOVED)
L18	1276	S (EICOSAPENT? AND DOCOSAHEXA?) AND FISH(W)OIL
L19	865	DUP REM L18 (411 DUPLICATES REMOVED)
L20	190	S L19 AND CHOLESTEROL